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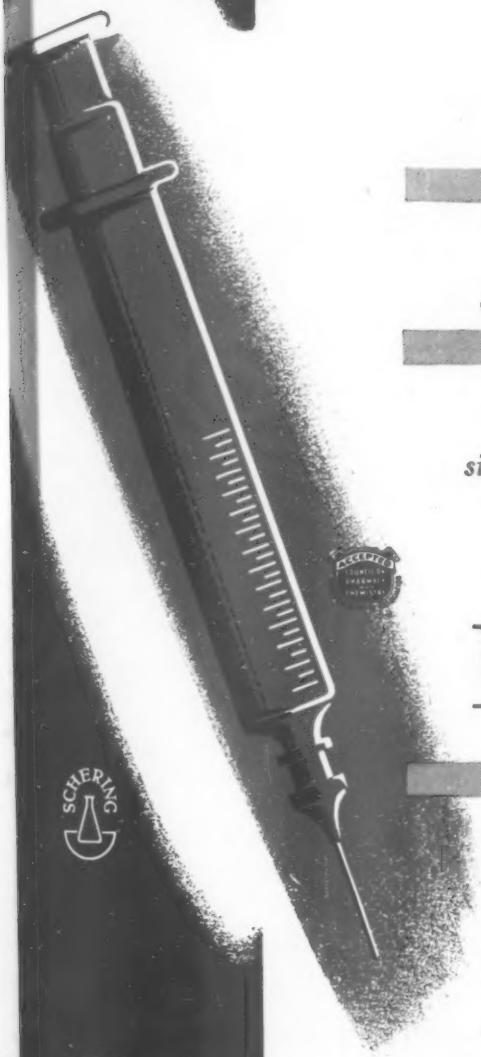

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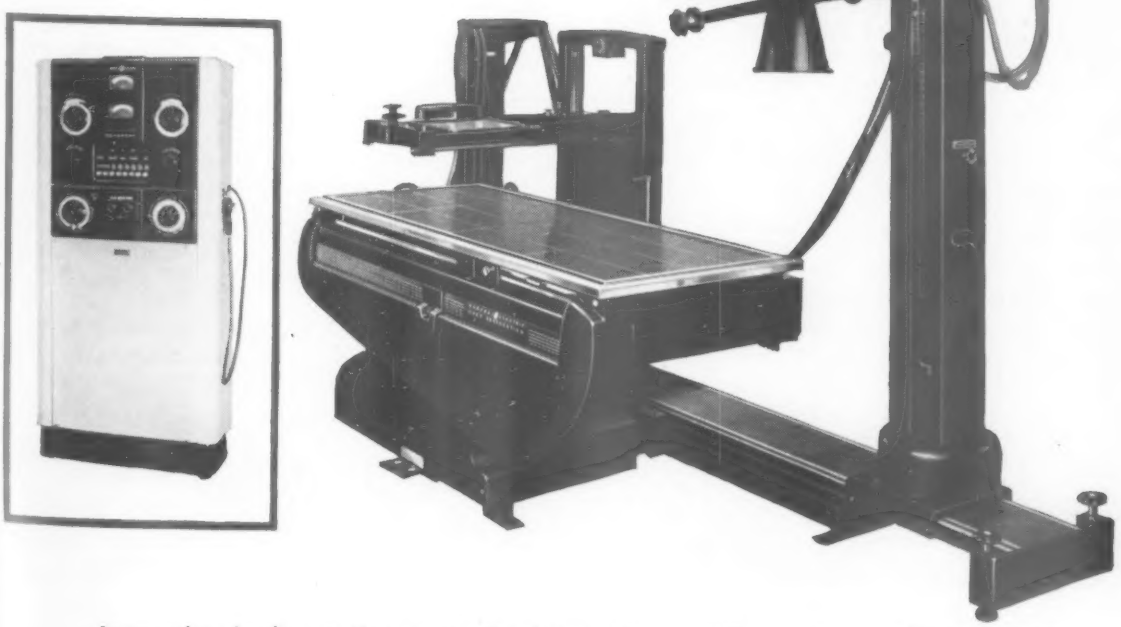

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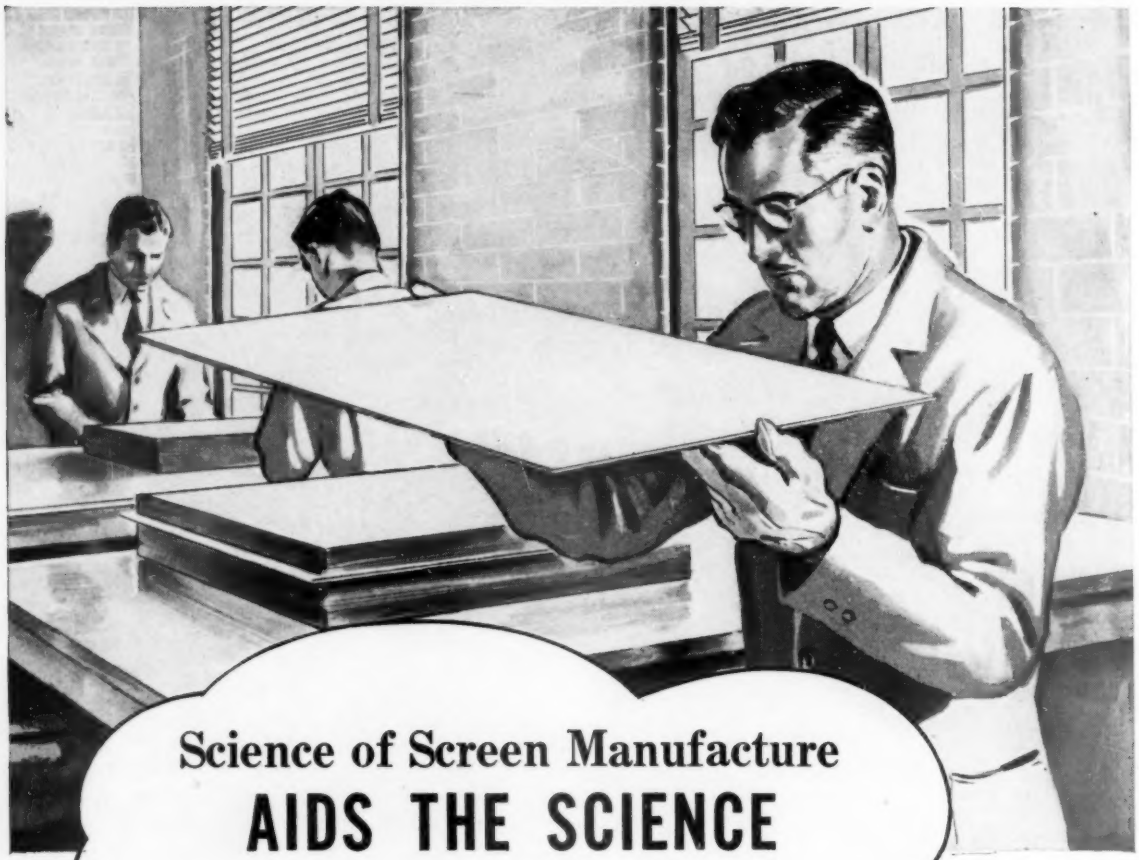

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THE ANATOMY AND PHYSIOLOGY OF THE LESSER CIRCULATION

AS INDICATED BY ITS BEHAVIOR IN HEALTH AND DISEASE*

By L. R. SANTE, M.D.

ST. LOUIS, MISSOURI

ANATOMISTS and histologists have learned much about the minute structure of the lung by patient examination of innumerable microscopic sections. Physiologists, by ingenious experiments, have done much to clarify our understanding of its function. There still remains, however, many ambiguous phases of the entire subject of lung structure and function.

The roentgenologist, in his wide field of clinical observations, has a means of testing the validity of these findings in practice, and of making suggestions for their modification. All of the lung structures are interdependent; any condition affecting one will have some correlated effect upon the others. The purpose of this paper, therefore, will be to seek out the conditions in which the roentgenological and pathological findings may be of value in clarifying such fundamental points. The structures to be considered and their influence on the lesser circulation are:

Interstitial tissue—a structural framework for the lung composed of,
Fibrous stroma
Elastic fibers
Respiratory tract—having an intact epithelial lining from the trachea to the alveoli
Blood vessels—two sources, bronchial from the systemic circulation, and pulmonary, constituting the lesser circulation
Relation of capillaries to alveoli
Normal respiratory function
Increased alveolar pressure—emphysema
Decreased alveolar pressure—atelectasis
Vascular engorgement—passive congestion from cardiac failure
Vascular occlusion—infarction
Nervous and muscular control of bronchi—bronchiectasis
Lymphatics—anatomical relationship and function
Edema of lungs—absorption of protein and water.

INTERSTITIAL TISSUE

The view taken by Macklin[19,20] seems most plausible. He thinks of the interstitial tissue as being a construction framework for the lung, extending from the hilum to the finest air cells, offering passage from the bronchi, blood vessels and lymphatics, and giving strength through its fibrous stroma

* From the Department of Radiology of St. Louis University School of Medicine and St. Louis City Hospital. Presented at the Forty-eighth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 16–19, 1947.

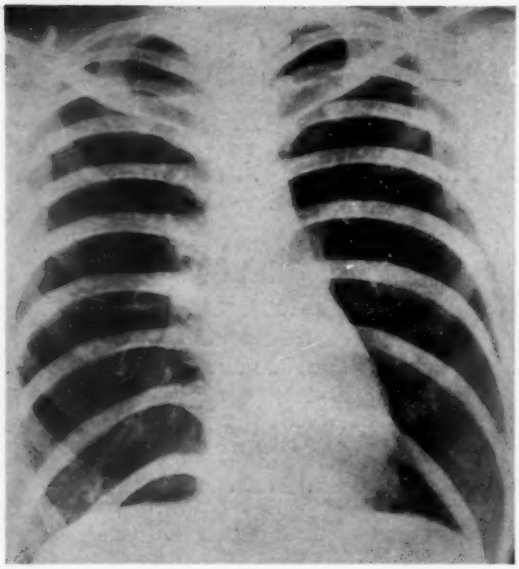

FIG. 1*a*. In a roentgenogram of the normal lung, the parenchymal structures show only in the peripheral zone. The terminal structures are so fine that the lung markings appear to feather out into imperceptibility.

for maintenance of the relationship of the other lung structures. The alveolar lining

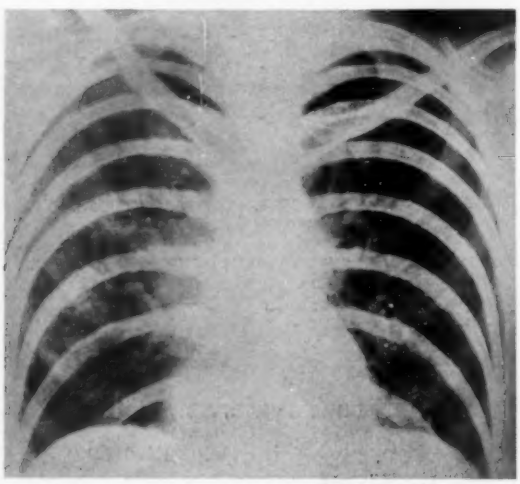

FIG. 1*c*. When the lung is involved by some disease which attacks the interstitial tissue, such as virus pneumonia, the existence of such interstitial tissue within the alveolar wall between epithelial layers of adjacent alveoli becomes quite evident. The infiltrative process seen in the peripheral zone of the roentgenogram is due largely to involvement of the interstitial tissue.

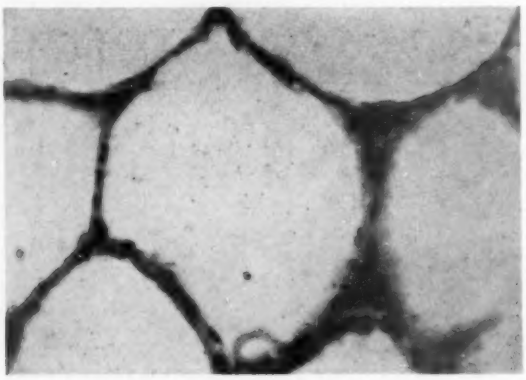

FIG. 1*b*. From the thinness of the mormal alveolar wall, it is hard to conceive the existence of interstitial tissue between the layers of alveolar epithelium of adjacent air cells.

is a continuous membrane of epithelial cells held in a close relationship to its surrounding capillaries and to each other by this fibrous tissue stroma in order to insure promptly coordinated reaction following changes in relationship of these structures. The stroma of the alveolar wall contains not only white fibrous tissue fibers but also yellow fibers of elastic tissue. "Stretching of the air sac walls is attended by a radial pull upon the vascular walls, and also by a longitudinal pull, just as with the walls of the air tract—bronchi, bronchioli and alveolar ducts. Deflation of the air space is accompanied by a recoil of the elastic tissue

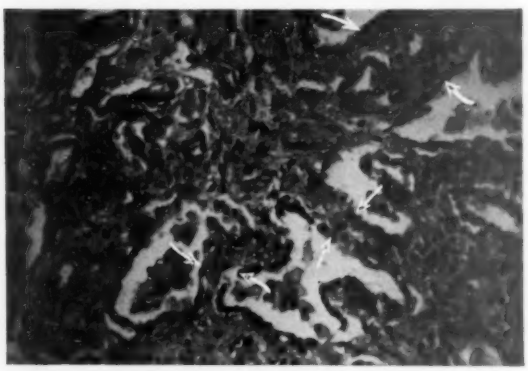

FIG. 1*d*. Contrast the enormous thickening of the alveolar wall in virus pneumonia due to interstitial involvement. Note two distinct layers of epithelium for adjacent alveoli with thick intervening interstitial tissue.

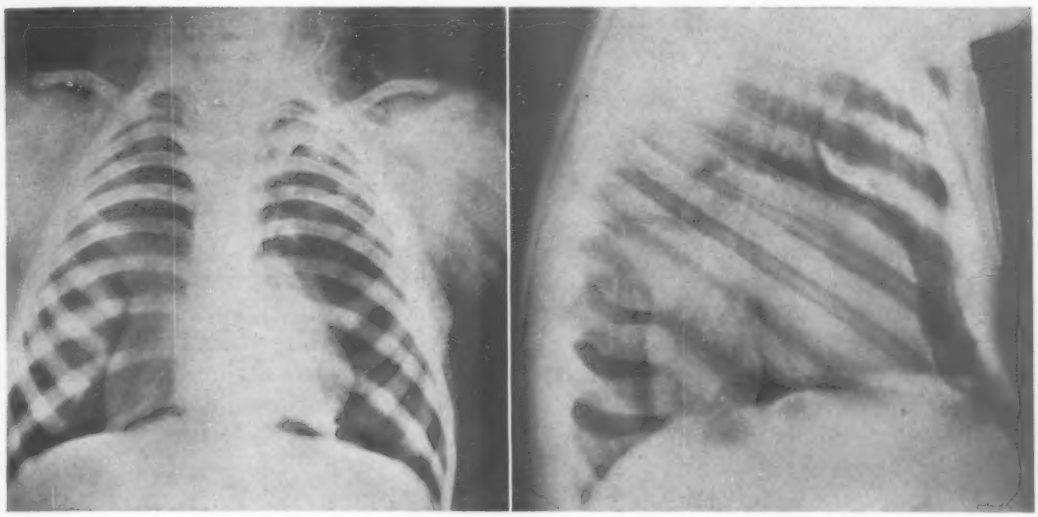

Fig. 2*a*. Anteroposterior and lateral roentgenograms of the chest. Air introduced into the mediastinum through a tracheotomy or other wound may be forced outward through the interstitial tissues of the lung to the finest lung structures, causing rupture of the alveoli, bilateral pneumothorax and death. This would indicate the existence of a continuous interstitial space throughout the lung.

and these spaces are all shortened and narrowed. . . . It is fortunate that the stroma is attached thus to the blood vessels, for otherwise there would not be this instant and favorable adjustment of their walls and calibre to the needs of the inspiratory phase."

It is true that the septum between adjacent alveoli (Fig. 1*a*, *b*), on microscopic examination seems hardly wide enough to represent two layers of epithelial cells, one for each alveolus, with intervening interstitial tissue for passage of blood vessels. Microscopic examination of sections of the lung, however, from patients infected with certain diseases, such as virus pneumonitis (Fig. 1*c*, *d*) which attack the interstitial tissue, shows marked thickening of the interstitial space with cellular infiltration, and inflammatory reaction of the intact alveolar cell membranes (Sante).[30]

In the hilar regions the interstitial tissue is large in amount offering passage for the bronchi, blood and lymph vessels; the bronchi are supported by cartilaginous rings, but the vascular structures are not so firmly constructed. The fibrous interstitial tissue about the vessels arranges itself into tough perivascular sheaths. That these perivascular sheaths are continuous, extending throughout the interstitial space from the hilum to the terminal air cells in one direction and back into the mediastinal space in the other, is indicated by the pathway traveled by air which gets into the interstitial tissue of the lung (Fig. 2*a*, *b*) (Macklin[18,21]). Interstitial emphysema may originate in the periphery from rupture of an emphysematous bleb (Sante[28]), extend-

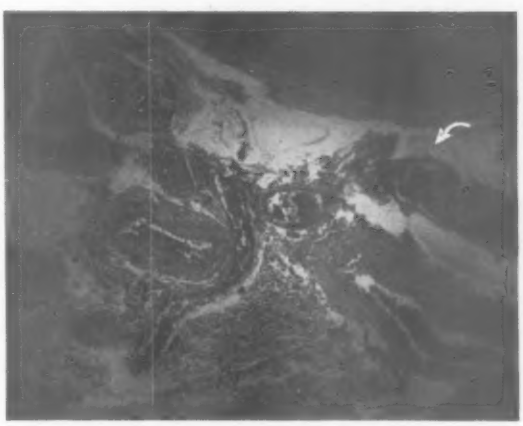

Fig. 2*b*. Microscopic examination shows air in the interstitial spaces surrounding the vascular structures, as it spreads to the periphery to the finest lung structures.

ing to the mediastinum and producing the appearance of a halo about the heart characteristic of air in the mediastinal space. Or the air may gain entrance into the mediastinum directly through an operative incision from tracheotomy or other wound. Large amounts of air sucked into the mediastinal space in this manner spread rapidly through the interstitial framework of the lung to the periphery causing rupture of the lung and pneumothorax. Microscopic examination shows the air in the perivascular spaces of the interstitial tissue. Pressure from this entrapped air upon the vascular wall interferes with circulation of the blood and may cause death. This is perhaps the most convincing proof of a continuous interstitial space from the periphery of the lung, back through the root of the lung and into the mediastinum.

RESPIRATORY TRACT

The gross anatomy of the lung is quite clearly understood (Brock[5]); the finer lung structures, comprising the minute anatomy of the lung, still require clarification on some points.

William Snow Miller's[23] microscopic study of the primary lobule of the lung is a classical foundation for further consideration and investigation. His observations will be recalled that, as the bronchi extend toward the periphery, they continue to divide and subdivide until they reach a point where the cartilaginous rings, which form a supporting framework for the bronchioles, disappear, leaving only the musculature and fibrous stroma for the support of the bronchioles. At this point in the bronchial subdivision the bronchioles have air cells opening off directly from their walls which prompts their designation as respiratory bronchioles. From this location the bronchioles divide and subdivide into ductulus alveolaris, atria, sacculi alveolares, terminating finally in alveoli pulmonum or terminal air cells.

The portion of the lung structure distal to the respiratory bronchiole may be considered as the parenchymal tissue of the lung. Roentgenological examination after the intratracheal injection of iodized oil will serve to trace these divisions of the bronchial tree to the minutest structures. This would indicate that there is an intact epithelial lining of some sort for the respiratory structures from the bronchi to the terminal air cells.

Some observers maintain that no separate alveolar epithelium exists and that the capillary walls of the blood vessels form the lining membrane of the alveoli contained in a ground substance directly exposed to the alveolar air, with occasional "septal cells" probably of mesenchymal origin (Lang,[15] Bloom,[4] Loosli[17]). Certain microscopic sections showing cuboidal cells extending part way around the alveolar membrane apparently gives support to this contention (Aschoff,[1] Seemann,[31] Bargmann[2]) but these are probably due to bronchioles cut obliquely. Others feel that the alveoli are formed by an intact epithelial lining and that the capillaries form an independent syncytial network covering them (Miller,[23] Bremer[3]). It is difficult to understand how the capillary walls could themselves form the lining of the alveoli and still present an intact circulation. This would imply a transition of bronchial to vascular epithelium at some point beyond the respiratory bronchiole. This seems hardly possible since Miller[23] has traced the bronchial epithelium peripherally to the terminal alveolar structure. Furthermore, he has demonstrated in microscopic section an intact ring of epithelial cells lining the alveolus. It is true that this was in an edematous lung which, in the strictest sense of the word, could not be considered as an absolutely normal structure, but such an argument seems illogical since no pathological lesion exerts less injury to cells, nor excites less reaction than transitory edema; it is, in fact, merely a physiological response to variations in fluid pressures within the lung. There is hardly any other condition which can develop more rapidly and subside more quickly with complete restoration of the lung to normal. In the case of

mechanical edema of the lungs (Moragues[24]), which developed immediately after rupture of one of the papillary muscles of the heart causing death within a few hours, time would hardly permit the proliferation of cells to form a complete alveolar lining. This is convincing although not absolute proof of the existence of a separate epithelial layer of alveolar cells. If the vascular epithelium formed the alveolar lining of adjacent alveoli on either side, it would imply the non-existence of interstitial tissue between the alveolar walls.

VASCULAR STRUCTURE OF THE LUNG

The respiratory bronchiole is an important landmark also in the pulmonary circulation. The blood supply of the lung is from two sources, the pulmonary and bronchial vessels. The pulmonary arteries, carrying venous blood to the lung for aeration, accompany the bronchi, dividing and subdividing as they extend toward the periphery, sending minute branches to the finest subdivisions. These minute vascular branches have been used by Miller,[23] in tracing the finest respiratory subdivisions to their termination. A corresponding system of pulmonary veins returns the aerated blood to the heart. *The bronchial arteries*,[23] carrying arterial blood, act as *nutrient vessels to the bronchi* and are imbedded in their walls up to the region of the respiratory bronchioles; at this point the bronchial and pulmonary circulations mix in a plexus of capillaries which cover the lung beyond this region; *nourishment of the parenchymal* tissue of the lung, therefore, comes largely from the *pulmonary vessels*.

RELATION OF CAPILLARIES TO ALVEOLAR STRUCTURE

The structure and arrangement of the capillaries in relation to the alveoli are of greatest importance. It is obvious that they must form a thin vascular network covering the alveoli and that they must be shielded, in some way, during deep inspiration from pressure of the distended alveoli, between which they lie. We know that during inspiration the volume of the chest cavity is much increased in size due to the action of the musculature of the thoracic walls and diaphragm (Best and Taylor[3]). This increase in volume is taken up both by an increase in air and blood content. It has been shown that the blood volume in the lung increases as much as 50 per cent during deep inspiration. This is a means of producing greatest aeration of the blood. In order for this to take place and for the thin-walled capillaries to withstand pressure from the distended alveoli there must be some structural safeguard. This is found in the elastic tissue which runs in strands within the fibrous stroma, like guy wires over the alveolar membrane.[23] The exact location of the elastic fibers is difficult to show microscopically but their function should serve to indicate their location. Their purpose is to limit and prevent overdistention of the alveolar structure and to offer recoil elasticity to the alveolar wall. Like elastic fibers in any location they offer more and more resistance as they undergo greater and greater distention. They are imbedded in the stroma and extend over the alveolar epithelium forming a part of the alveolar wall. The capillary network covers the alveoli intervening between adjacent alveolar structures. The fibrous stroma covering the capillaries holds them in intimate relationship to the alveolar walls but does not contain elastic tissue elements. The elastic fibers could not extend over the capillary network since if this were true alveolar distention would produce vascular constriction and thus prevent maximum aeration during inspiration.

Normally, the small venules in the lung which supply the capillary network are also closely surrounded by the alveoli; in fact, the alveoli which abut on the walls of the venules serve to aid in the support of the vascular wall—the amount of intervening interstitial tissue is very small. It is obvious then that any change in size of the surrounding alveolar structure or any change in the interalveolar pressure will be transmitted directly through the thin-walled

vessel, thus helping to equalize any changes which may take place between the alveoli and the circulation.

Macklin's observation,[19] "because the expansile force of the incoming air is spread by means of the perivascular sleeve of air cells, over the arterial and venous systems rather than being concentrated upon the pulmonary capillaries . . . it is felt that the capillaries are protected from possible harm," seems quite valid. That the "expansile force of incoming air" is not an independent force acting upon the capillary walls, however, but rather a passive response to the negative pulmonary pressure producing it, which acts *alike* on both *capillaries and alveolar walls*, must not be overlooked. The gradient of force acting upon the air cells, and capillaries concerned in carrying on the function of the lung, has been fully discussed by Wasson.[32]

The alveolar walls are guarded from overexpansion by elastic fibers whereas the capillaries have only a fine fibrous stroma. The increase in blood volume in the lung during deep inspiration is explained by the fact that the *blood capillaries* thus guarded from pressure by overdistention of the *alveoli are themselves subject to changes in intrathoracic pressure* so that the development of a negative pressure in the thorax during inspiration produces greater vascularity as well as greater engorgement of the blood capillaries in contact with the alveolar membrane, all desirable effects in aeration of the blood. The pull of the stroma maintains the close relationship of the capillaries to the alveolar walls and aids in their expansion and elongation. On *expiration*, pressure of the distended alveoli on the capillaries squeezes out the excess blood, thus producing a pump-like action which is a very potent factor in aiding the pulmonary circulation.

Roentgenographically, the increased illumination of the lung field during deep inspiration indicates the relatively greater percentage of air-to-blood content in the lung. Westermark[33] has shown roentgenographically the effect of pressure on the capillaries of the lungs while in their fully inflated state, by the Valsalva exercise (forced expiration against obstruction). Under such circumstances the air pressure in the distended alveoli is increased by attempting forced expiration against the closed mouth and nose; the marked blanching of the lung field as shown on roentgen examination during this procedure is indication that the capillaries are subject to further action in expressing still more blood from the vascular structures by thoracic pressure.

This would indicate the close relation of the blood capillaries to the alveolar structure of the lung; their prompt response to changes in alveolar function; the protection of the alveoli by elastic fibers within the alveolar walls, and of the blood capillaries by the spread of the forces exerted by alveolar changes to all of the vascular structures.

Increased Alveolar Pressure—Emphysema. Let us consider the effect of increasing alveolar pressure as in pulmonary emphysema (Fig. 3*a* and 3*b*). One may judge the function of the elastic tissue on the lung structure by considering the effect produced in pathological conditions in which it is congenitally absent or destroyed. Emphysema is probably produced by defects in the elastic tissue structures either inherent or acquired. In this condition the restraint which is normally present on alveolar distention is removed, the alveolar pressure rises and the alveoli become enormously enlarged and overdistended even to the point of rupture. Likewise, the elastic recoil of the lung is impaired so that the air content is never properly expelled. As a result, the distended alveoli squeeze the capillary network, preventing proper circulation of the blood, with resulting dyspnea and cyanosis.

Roentgenographically, this is indicated by increased radiolucency of the lung, but little if any change in illumination during inspiration or expiration. The Valsalva exercise likewise does not show any change in the illumination of the lung fields, indicat-

ing that there is very little displacement of blood from the already impoverished pulmonary circulation. The pressure of the distended alveoli likewise is exerted upon the smaller bronchioles interfering with all but the slightest passage of air to the alveolar structure. As a result, it is seen that iodized oil injected into the bronchial tubes will not pass out into the alveolar structure, but remains confined to the larger bronchial branches (O'Donoghue[28]).

Fig. 3*a*. The best way to demonstrate the function of the elastic tissue fibers in the alveolar wall is to study the behavior of the lung where these fibers are deficient. In emphysema the intra-alveolar pressure becomes increased due to failure of elastic tissue to express the air on expiration; the unrestrained distention of the alveoli during inspiration squeezes the unprotected intervening capillaries and as a result there is not only enormous dilatation and rupture of the alveolar walls but marked avascularity of the lungs as well. Lipiodol injected into the emphysematous lung does not proceed down into the alveolar structure due to the increased intra-alveolar air pressure.

Emphysematous bullae, formed by overexpansion of the alveolar structures from the check-valve action produced by pressure on their ducts may even be outlined by the oil-filled bronchioles; the oil does not enter the bullae. Within twenty-four hours the oil will be expelled from the emphysematous lung. Normally, iodized oil injected in this way will pass out into the alveolar structures producing a feathery appearance on roentgen examination which will remain for many months. *In obstructive emphysema*, however, the overdistended alveoli have their elastic fibers intact, merely squeezing blood out of the lung by their dilatation as shown roentgenographically by hyperaeration of the lung and return to normal after relief of obstruction. In this respect compensatory emphysema differs from emphysema of the hypertrophic type.

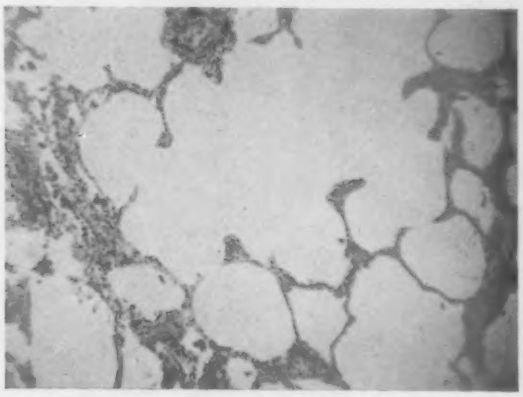

Fig. 3*b*. Microscopic examination in pulmonary emphysema reveals distention, or breakage through, of the alveoli from increased intra-alveolar pressure. Unrestrained pressure within the alveoli causes almost complete avascularity of intervening capillaries.

Decreased Alevolar Pressure—Atelectasis. In normal inspiration the lung expands passively by an inrush of air, as a result of enlargement of the chest cavity; the negative force is exerted from *outside* the alveolus and the inrush of air occurs to equalize the pressure. If a bronchus is completely occluded by a plug of mucus, foreign body, or from other cause, the peripheral lung segment will become atelectatic and collapse from absorption of the entrapped air. In atelectasis, then, the process is reversed; the suction force produced by absorption of alveolar air is exerted from *within* the alveolus (Fig. 3*c* and 3*d*). Diffusion of gases through the alveolar membrane continually takes place in order to equalize the atomic pressure. The capillary circulation continually offers a fresh supply of blood for aeration whereas the alveolar air is blocked in

the alveolus and cannot be renewed. The suction force thus created within the alveoli is tremendous. Elastic tissue prevents overdistention of the air cells in maximum inspiration but there is nothing to restrain them from complete collapse. Just how does the pulmonary circulation fare under such conditions? We have seen that the capillary network is almost directly exposed to all changes in intrathoracic pressure; only the slight resistance from the capillary wall is interposed. The pumping action of respiration is suspended; the sucking action of air cells produced by continuous absorption of the alveolar air is exerted directly upon the vascular walls also; both are factors in producing engorgement of the circulation. In this instance the force is so great that the chest wall is drawn down, the diaphragm displaced upward and the mediastinal structures pulled over to the affected side. Such force directly applied to the walls of the vascular organs of the lungs should be very potent in producing an engorgement of the lung vessels, especially of the capillary structures. The extreme density of the lung field (as dense as lobar pneumonia) is evidence of vascular engorgement. The prompt disappearance of the lung consolidation with reinflation of the lung, after rolling the patient upon the unaffected side, is added indication that vascular engorgement is responsible for the increased lung density (Sante[27]).

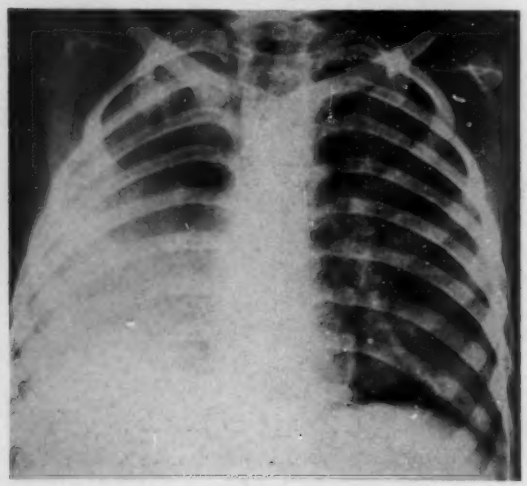

FIG. 3*c*. Decreased intra-alveolar pressure occurring in atelectasis results in a sucking action exerted on the alveolar walls. The alveoli are protected by elastic tissue from overdistention but there is no action in protecting against excessive deflation. As a result, there is engorgement of the capillaries and other blood vessels of the lungs. It is this vascular engorgement which imparts to the atelectatic lung the extreme roentgenographic density. The engorgement occurs in passive response to the sucking action of decreased intra-alveolar pressure and therefore there is little if any edema of the lung.

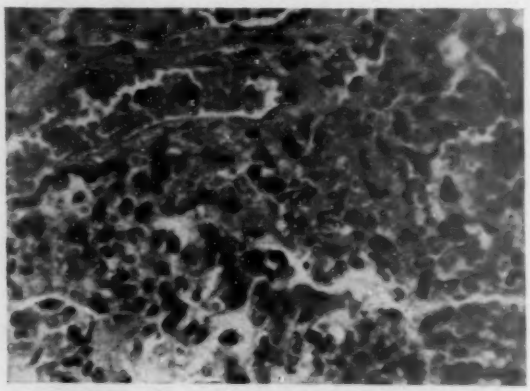

FIG. 3*d*. Microscopic section of pulmonary atelectasis. Note collapse of alveolar walls, widening of interspaces between alveoli, and marked capillary engorgement.

Roentgenological as well as clinical evidence would indicate that there are two forms of massive atelectatic collapse of the lung, the one due to bronchial occlusion by a single gelatinous inspissated plug of mucus, the other resulting from profuse secretion which may have its origin as an exudate from the parenchymal structures, the so-called "drowned lung;" both causing atelectasis because of bronchial occlusion from loss of the cough reflex. In the dry type, re-expansion takes place immediately, within a few minutes, following rolling the patient on the uninvolved side and the expectoration of a single "glob" of mucus which "stands up like a gumdrop on the bottom of the cup;" in the "drowned lung" type re-expansion takes place more slowly; the patient coughs up greater quantities of thinner sputum before re-expansion takes place.

If a single lobe is involved, the sucking action produced by the atelectatic process is transmitted to the adjacent aerated lung causing compensatory emphysema. It is interesting to note, by lipiodol injection, that the suction force is not equally divided throughout the lung but is most pronounced in the immediate vicinity of the collapsed lobe.

We know that the healthy alveolar membrane will also permit air to pass directly through from one to another adjacent lobule, since air injected into one bronchiole can readily be demonstrated coming out of an adjacent bronchiole of the same lobe. Small openings or pores between adjacent alveoli have been demonstrated microscopically (Miller[23]). This mechanism helps to equalize the pressure in adjacent air cells. One would think, therefore, that segmental collapse (atelectasis) of small portions of the lung would be impossible since air deficiency would be replaced from adjoining alveoli. But we know that segmental collapse does occur, so that our only conclusion must be that with the development of the slightest edema or infiltration this permeability of the alveolar membrane is lost. This is as it should be since the greater the distention of an alveolus and the greater the pressure of its air content, the more pervious it should become. We know of course that the greatest degree of aeration of the blood takes place when the alveoli are distended with air during inspiration and that, conversely, the more collapsed the alveoli, the more reduced the alveolar pressure; this should favor greater obstruction to the passage of air between the alveolar walls. Partial obstruction of a segment of the lung may result in segmental emphysema (Golden[12]).

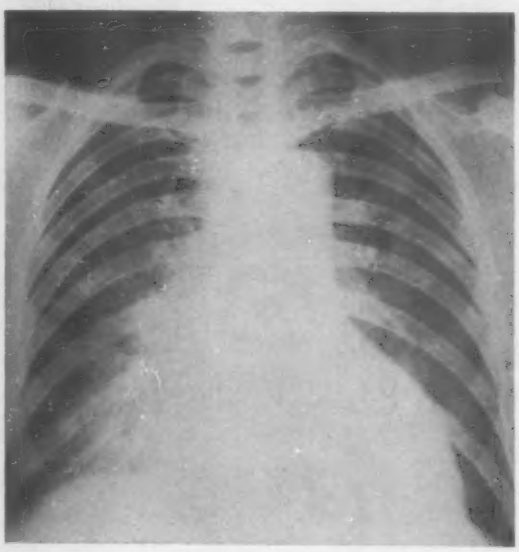

Fig. 4*a*. Passive pulmonary congestion produces in the roentgenogram a more or less characteristic picture: engorged vascular trunks radiate fan-shaped outward from both hilar regions into the periphery of the lung. The outer parenchymal zone is clear, however.

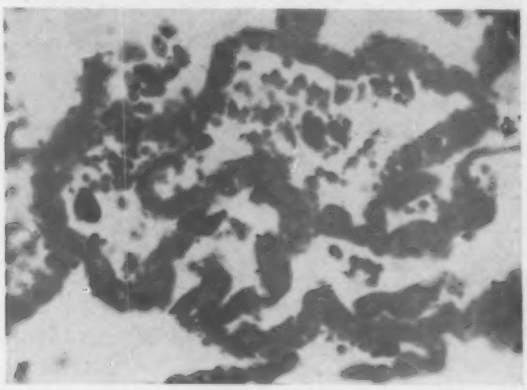

Fig. 4*b*. Microscopic findings in passive pulmonary congestion shows the enormously distended capillaries between the alveolar walls without evidence of intra-alveolar edema.

Vascular Engorgement. Passive congestion of the lung produces engorgement of the capillaries due to failure of cardiac action (Fig. 4, *a*, *b*, *c*, *d*). Stasis results and the capillary network becomes packed with red blood cells but they remain within their capillary walls as long as compensation is maintained.

Roentgenographically the stage of passive pulmonary congestion results in an increase in blood vascular markings radiating outward from the hilar region into the lung.

Pulmonary Infarction. Blood clots orig-

FIG. 4*c*. Edema of the lungs results when the pressure within the blood vessels exceeds the interalveolar pressure and serum exudes from the blood vessels into the alveoli.

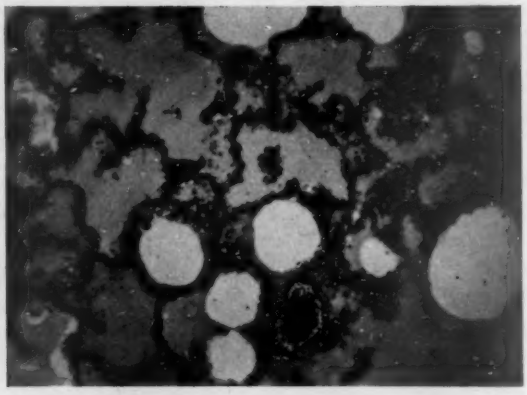

FIG. 4*d*. Microscopically edema of the lungs shows the fluid within the alveolar spaces. This cuts off the blood from aeration, producing an ever-increasing degree of anoxemia.

inating in the systemic circulation or heart may lodge in the pulmonary circulation forming emboli (Fig. 5*a* and 5*b*). Such emboli are not visualized in the roentgenogram since they are of the same density as the blood stream. Westermark has shown, by utilizing the Valsalva exercise, that blanching of the obstructed region occurs. If collateral circulation is adequate for nutrition of the structures, true infarction may not take place and the tissues may be restored to normal. Blood supplied by anastomosis with the adjacent capillaries and by the bronchial artery may be sufficient to prevent the death of the tissues. The bronchial artery is peculiarly adapted to this purpose; it arises from the systemic circulation (the aorta) so that it is not likely that anything should happen to it at the

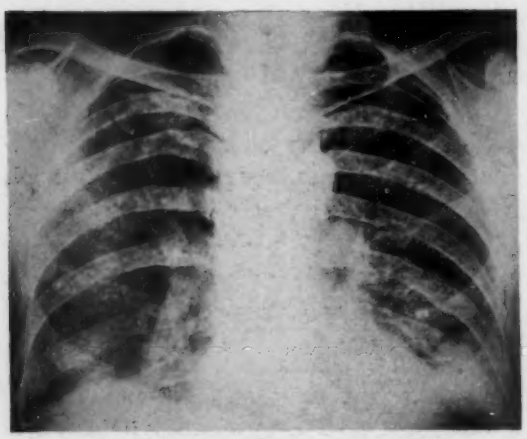

FIG. 5a_1. Infarcts of the pulmonary circulation have a tendency to occur at the periphery of the lung where the planes of the pleura form an angle with each other. These become consolidated, showing in the roentgenogram as areas of increased density, margins perpendicular to the pleural plane, inner edge may be rounded and smooth.

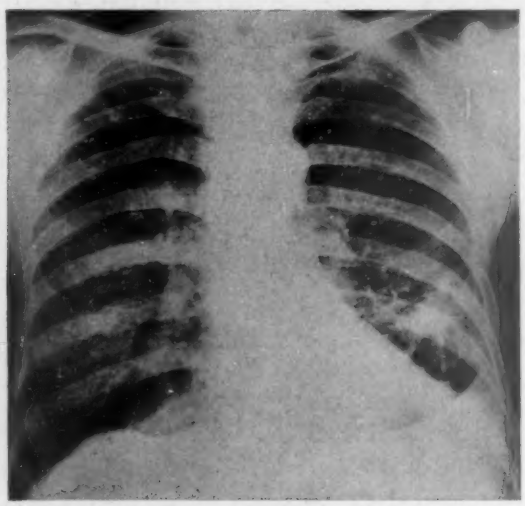

FIG. 5a_2. Organization of infarcts commences almost immediately. Even within a few days microscopic evidence of beginning organization is present; complete organization requires several weeks.

same time that infarction of the pulmonary circulation occurs. If the blood supplied from these sources is not sufficient to maintain the nutrition of the lung structure in the involved area, infarction occurs.

Then, and then only, does the infarct become visible in the roentgenogram. It has been found that infarcts usually occur at the periphery of the lung when two planes of the pleura meet the infarcted area showing a medial rounded border.[13] Just why infarcts occur in such regions is not definitely known but it probably is due to some particular arrangement of the vascular structures in these locations.

When an embolus lodges in the pulmonary vessel, the blood supply to the pa-

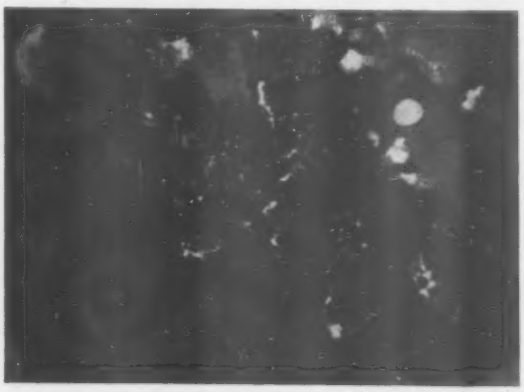

Fig. 5*b*. Hemorrhagic infarct of the lung. The lung is one of the few organs of the body provided with two circulations, the pulmonary and the bronchial. When the pulmonary circulation is blocked the bronchial artery pours blood into the area from the systemic circulation providing nutrition for the infarcted area.

renchymal structure from that source is cut off, and the area that it supplies is dependent upon blood borne by the bronchial artery and the collateral capillary circulation. Stasis occurs and blood exudes from the capillaries into the lung structure, producing a hemorrhagic infarct. Organization by scar tissue formation begins almost at once and is evident microscopically within a few days.

Unless the infarct becomes infected it rarely undergoes necrosis.

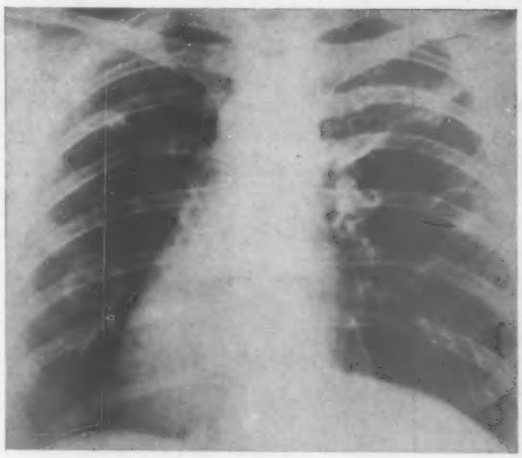

Fig. 5*c*. Infarcts involving the bronchial artery as well as the pulmonary vessels, caused by infection having a predilection for blood vessels, cause thrombosis, result in formation of nodules at the periphery of the lung which rapidly undergo central liquefaction producing thick-walled annular shadows of unusual type.

Thrombosis, on the other hand, resulting from infection by an organism having a predilection for vascular structures, may result in complete occlusion of the bronchial circulation as well (Fig. 5*c* and 5*d*) (Sante and Hufford[29]). Under such circumstances necrosis rapidly follows, even in a matter of hours. Rounded nodules varying from 0.5 to 3 cm. in diameter appear

Fig. 5*d*. Thrombosis of bronchial as well as pulmonary vessels from infection. This results in rapid formation of unusual annular shadows throughout the lung.

throughout the lung fields; these rapidly liquefy in the center leaving smooth annular shadows having thick but uniform walls.

These probably represent the line of demarcation between viable and destroyed tissue of the infarcted lobule. They may become thinner and thinner and ultimately disappear, or may remain evident in the roentgenogram for more than a year later.

FIG. 6*a*. In bronchiectasis the bronchial dilatation is limited to the distribution of a bronchus usually involving an entire lobe. In the roentgenogram the dilated bronchial branches extend peripherally to a point which would correspond to the respiratory bronchiole; the zone of normal appearing lung beyond is probably due to compensatory emphysema of the adjacent normal lung.

Microscopic sections show thrombosis in all of the vessels with evidence of an infection in the surrounding tissue.

Nerve and Muscular Control of Bronchi—Bronchiectasis. Bronchiectasis is primarily a disease of the smaller bronchi (Fig. 6). A consideration of the vast amount of experimental investigation, pathological and clinical observation (Lisa and Rosenblatt[16]) which has been carried out on the subject would indicate that bronchiectasis is caused primarily by severe infection of the bronchial wall. The mucosa is stripped up and the bronchial wall thus denuded is subjected to extension of the infection to the other bronchial structures: the muscle wall, elastic tissue, nerve fibers, and the blood vessels. Irregular areas of necrosis occur in these tissues, severing their continuity and thereby rendering them functionally useless. These have always seemed to me to be the essential factors in development of bronchiectasis. During normal respiration, the bronchi become larger in caliber and longer during inspiration, and smaller and shorter during expiration. This is due to the influence of neuromuscular control; muscle tone maintains the normal configuration of the bronchus due to the action of the same structures. In bronchography it has been noted that cocainization of the bronchus produces marked dilatation, and removes the natural diameter changes ordinarily observed in normal bronchi during respiration. These are exactly the same conditions which prevail in bronchiectasis, the bronchial wall loses its tone, dilates and no longer responds to respiratory changes. Add to this infection which has invaded and destroyed areas in the bronchial wall and

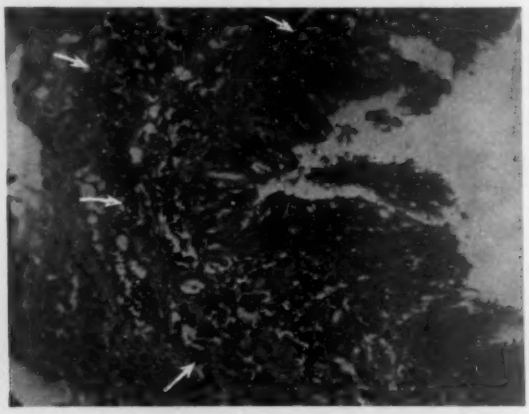

FIG. 6*b*. Microscopic examination in bronchiectasis shows destruction of bronchial mucosa, with undermining and stripping up of epithelium by infection; and destruction of the bronchial musculature, thus removing its support from the bronchial wall and permitting dilatation of the bronchus.

the picture is practically complete. There still remains the alveolar structure to be accounted for; edema alone, resulting from such profound infection, would be sufficient to cut off the parenchymal structure supplied by the involved bronchus. Atelectasis of such a small volume of lung tissue followed by fibrosis would hardly be detectable in the roentgenogram. As this small volume of atelectatic or scar tissue contracts, the space is filled in from either side by compensatory emphysema from the adjacent alveoli. As the acute infection subsides the areas of destruction in the bronchial wall heal with fibrous tissue formation and the non-flunctioning state of the bronchial musclature becomes permanent. Epithelization of the chronically infected bronchus completes the picture.

Roentgenographically, lipiodol readily enters and outlines these blind bronchial pouches and shows they have no muscular tone. The failure of any alveolar filling to occur from these bronchi, and the ease with which they are emptied, indicates that their parenchymal lung tissue is destroyed. The distance observed between the end of the blind pouch and the pleural surface indicates the size of bronchus involved by the process.

The Lymphatic Structures of the Lung. Again the region of the respiratory bronchiole is an important spot. From this point the lymphatic drainage of the lung progresses in two directions, one toward the hilum by the lymphatic trunks which accompany the vascular system, the other toward the periphery of the lung to the pleura, over its surface terminating likewise in the hilar region. Valves in the lymphatic vessels prevent reverse flow. The smallest lymphatic divisions do not extend to the alveolar structure but end at the alveolar ducts. The function of the lymphatics is to absorb any proteinized fluid which may escape from the vascular structures and return it to the blood stream, thus guarding against its loss to the body; likewise, to absorb any form of foreign material such as coal dust or bacteria, filtering them out by their glandular structures, as pointed out by Drinker[10]. The lack of lymphatic structures peripheral to the alveolar ducts would indicate that the alveolar membrane would have to become extremely swollen and edematous and fluid would have to exude into the alveoli before it would be subject to absorption by the lymphatics. Such fluid may be disposed of either by coughing and expectoration, in which case it is lost to the body, or by absorption of the protein either in its unchanged state or after being broken down by enzymes, in which case it is restored to the body. Krogh[14] has pointed out that anoxia increases the permeability of the capillaries. There is a loss of blood volume with concentration of corpuscles in the capillaries and a reduced volume flow which further reduces the delivery of oxygen to the tissues and thus a vicious circle is established. Krogh further states that the capillary status resulting from oxygen deficiency is irreversible after fifteen minutes. As Drinker[9] appropriately points out, "anoxia begets anoxia."

A case reported by Moragues[24] of spontaneous rupture of a papillary muscle of the heart affords a good example of purely mechanical acute engorgement of the lung. The patient was stricken while eating his lunch and survived only a few hours. Autopsy revealed extreme vascular engorgement and extreme edema of both lungs.

It is evident that pulmonary edema, if it occurs in any sizable area of the lung may become very dangerous; this is especially significant in surgical cases and should be guarded against by all possible means (Chase[7]).

As Drinker[10] points out, one would expect to find large lymphatic vessels draining into the thoracic duct so that there would be the freest possible movement of the lymph toward the great veins but that no such arrangement usually exists; instead, all lymph from *both lungs*, except for a small area in the left apex, drains by way of the right lymphatic duct into the right subclavian vein. This right lymphatic duct

"is an exceedingly short and small vessel, and is thus a definite bottleneck at the end of a widespread system of lymphatic vessels."

If the pulmonary vein is obstructed, there is an immediate increase in the lymph flow from the right thoracic duct, indicating that there is an immediate transudation through the capillaries into the tissues and then into the lymphatics. Passive pulmonary congestion associated with cardiac decompensation results in similar retardation of blood flow and produces similar results. The lymphatics protect the body from loss of protein substance; any inadvertently lost in the tissues is absorbed by the lymphatics and returned to the circulation by the subclavian vein.

The fluid and electrolyte balance between the tissues and the vascular and lymphatic structures is maintained by the hydrostatic osmotic pressure on either side of the permeable membrane. Drinker[10] points out that " . . . in any consideration of a balance of forces the membrane itself must never be forgotten; and when the membrane is living, it may vary in permeability almost as rapidly as hydrostatic pressure change, and infinitely more rapidly than alterations occur in concentration of blood protein, upon which the colloid osmotic pressure depends. It is my belief—that simple pulmonary edema and the more serious pulmonary exudations depend more upon alterations in the permeability of the lung capillaries than upon complicated pressure relations in the pulmonary circulation." Just how great an influence the nervous system has in regulating the permeability of these walls is not known but extensive research would indicate that there is a definite regulatory mechanism (Bülbring and Whitteridge[6]). The colloid osmotic pressure is about 25 to 30 mm. of mercury throughout the systemic as well as the pulmonary circulation, but the capillary blood pressure in the lungs is about one-half to one-third that present in capillaries of the body tissues elsewhere. Whereas the body tissue should be just on the verge of spilling fluids into the tissues, the lung tissues should show greater assurance of a dry lung.

This would also explain the pronounced absorptive powers of the normal lung. Water aspirated in to the lung is rapidly absorbed *by the capillaries* provided there is an intact circulation; it does not result in increase in lymphatic flow (Courtice and Phipps[8]).

Water is an essential substance for the continued normal function of the body. The chemical composition of fluids contained in various structures of the body differs greatly. Muether[25] states, "It is obvious that the plasma and interstitial fluids are practically identical except for the amount of protein which is much higher in plasma than in interstitial fluid. This indicates that the extracellular electrolytes can be easily pass to and from the intravascular and interstitial fluid. . . . Exchange between the blood and the interstitial fluid is accomplished primarily by variations in the capillary blood pressure and protein content of the interstitial fluid, on the one hand, and the tissue tension and serum proteins, on the other."

The mechanism of thirst usually serves to maintain the fluid balance under normal conditions; under certain abnormal circumstances, however, it may be necessary to utilize other parenteral methods for administration; subcutaneous, intravenous, intracavitary, or into the marrow cavities of bones (Muether[25]). All of these are subject to the regulatory mechanism of absorption except the intravenous method which not only dumps the increased load of fluid upon the circulation but serves to dilute the blood and disturb the fluid balance.

While the danger of overhydration is looked upon lightly by the surgeon, the roentgenologist sees many cases in which too rapid administration of large amounts of fluid results in spilling over of fluid into the interstitial and alveolar structures of the lung. The shifting of such edematous areas to the most dependent portion of the lungs by change in position of the patient is indication that dependency is a factor in

its production. Many factors enter into the development of such a "spill over" from the circulation which are fully appreciated and can be avoided (Elman,[11] Marriott[22]).

To recapitulate: The lymphatics do not surround the alveolar structures like the blood capillaries; the right lymphatic duct drains the lungs but is inadequate in most instances to take care of excess quantities of exudate rapidly forming, thus producing a bottle-neck in the flow of lymph from the lung. This would indicate that the regulation of fluid balance in the lung structures is a complicated mechanism depending upon the water-electrolyte balance between the capillaries and surrounding tissues and the state of the intervening permeable membrane. The lymphatics have to do with the absorption of protein which is spilled over into the tissues and its return to the blood stream. Excess water, on the other hand, is readily absorbed by the capillaries.

SUMMARY

The principal function of the lung is the aeration of the blood; the two structures concerned in this function, the blood-containing capillaries and the air-filled alveoli, come into relationship in the terminal air cells. All of the anatomical arrangements here described are concerned with the fulfillment of this function.

Gaseous interchange takes place between the alveolar air and the blood by diffusion of gases through the alveolar lining and capillary wall. This is the process which takes place with each normal respiration. Anything which interferes with this normal mechanism will have an influence on the respiratory function.

The behavior of the respiratory function under various pathological conditions involving the lung structures has been described and discussed. A knowledge of the microscopic pathology should aid in the understanding of the roentgen manifestations produced by these diseases.*

606 Missouri Bldg.
St. Louis 3, Missouri

* For discussion see page 37.

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Vol. 61, No. 1

PERIPHERAL VASCULAR DISEASE IN THE LUNGS*

By ROBERT P. BARDEN, M.D., *and* DAVID A. COOPER, M.D.
PHILADELPHIA, PENNSYLVANIA

AN ATTEMPT has been made in previous papers[1,2] to outline the pathologic conditions which produce changes in the peripheral blood vessels of the lung and to illustrate the roentgen appearance of the chest in some of them. The radiologist should recognize that vascular disease may produce characteristic shadow patterns which differ from those which accompany disease of the lymphatic or alveolar pulmonary structures.[3] Furthermore, conditions which produce obstruction or obliteration of peripheral vessels may be distinguished from those accompanied by increased permeability of the vessel walls. However, a clear understanding of the pathology and clinical features of the group of diseases in question (Table 1) is essential, and serial roentgen examinations are often necessary before an accurate differential diagnosis can be made.

TABLE I

SOME CONDITIONS WHICH MAY PRODUCE CHANGES IN THE SHADOWS OF THE PERIPHERAL VASCULAR SYSTEM IN ROENTGENOGRAMS OF THE CHEST

1. Vascular Congestion	
	Heart failure, congenital heart disease, hemangioma
2. Intrinsic Obstruction of Blood Vessels	
	Thrombosis and embolism, trauma, polycythemia, leukemia, parasites, tumor emboli
3. Intrinsic Disease of Blood Vessel Walls	
	Sepsis, syphilis, arteriolar sclerosis, rheumatic fever, scleroderma
4. Obliteration of Vessels by Adjacent Pulmonary Disease	
	Pneumonia, tuberculosis, silicosis, carcinoma, abscess, emphysema
5. Hypersensitivity States and Toxins Causing Increased Permeability	
	Periarteritis nodosa, exfoliative dermatitis, influenza, lupus erythematosus, glomerulonephritis, eclampsia, beriberi, sulfa poisoning.

ILLUSTRATIVE CASES

1. *Congestive Lesions.*

Pulmonary hemangioma.

CASE 1. A. L., female, white, aged fifty, complained of cyanosis and chronic cough with occasional hemoptysis for many years. Examination showed multiple subcutaneous hemangiomas, most prominent over the dorsum of each hand. Hemoglobin 110 per cent, red blood cell count 7,000,000, white blood cell count and differential normal. Bone marrow smear showed nothing abnormal. Serial roentgenograms of the chest revealed unchanging nodular shadows scattered through both lower lung fields which were thought to represent pulmonary hemangiomas (Fig. 1).

FIG. 1. Case 1. Roentgenogram of the chest of a patient with dyspnea, cyanosis and polycythemia (probably secondary type). The multiple nodular shadows in the lower lung fields were thought to represent hemangiomas because of the associated extensive cutaneous hemangiomas which the patient presented. Note the irregular shape of the nodules and the lack of a sharp border.

* From the Department of Radiology and the Department of Medicine, Hospital of the University of Pennsylvania, Philadelphia.
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Comment. Although final confirmation is lacking in this case, the association of the pulmonary shadows with the polycythemia and subcutaneous hemangiomas make a diagnosis of pulmonary hemangiomas tenable. Additional proof might have been obtained by careful roentgenoscopy which should show pulsation of the nodules and diminution in their size when the patient performs forced expiration against the closed glottis.[9,10]

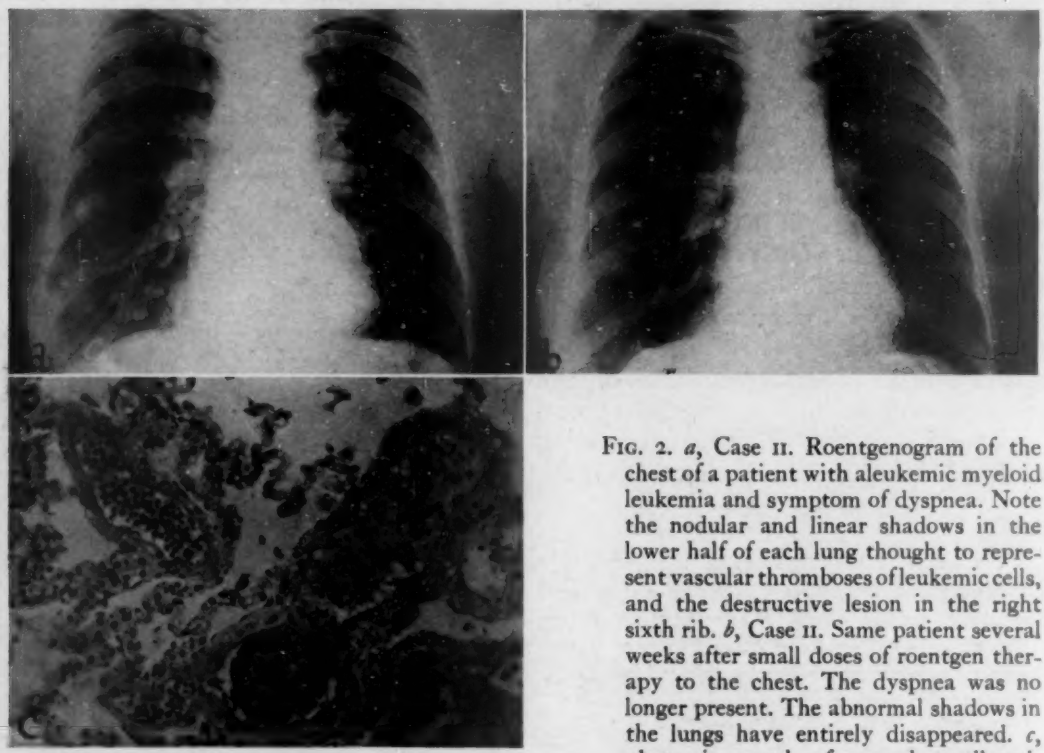

FIG. 2. *a*, Case II. Roentgenogram of the chest of a patient with aleukemic myeloid leukemia and symptom of dyspnea. Note the nodular and linear shadows in the lower half of each lung thought to represent vascular thromboses of leukemic cells, and the destructive lesion in the right sixth rib. *b*, Case II. Same patient several weeks after small doses of roentgen therapy to the chest. The dyspnea was no longer present. The abnormal shadows in the lungs have entirely disappeared. *c*, photomicrograph of several small pulmonary blood vessels from a patient with a similar history. In the lower center is a vessel with an organized thrombus containing degenerating leukemic cells. At one and eleven o'clock are vessels cut sagittally, containing excessive numbers of leukemic cells and probably undergoing early thrombus formation.

2. *Intrinsic Obstruction of Pulmonary Vessels.*

Leukemia.

CASE II. M. T., female, white, aged fifty-two, complained of enlarged lymph nodes, subcutaneous nodules, anema, and purpura of two years' duration. Intermittent cough and dyspnea were relieved by roentgen therapy. The peripheral white blood cells were normal, but the bone marrow smear showed myeloid leukemia. Roentgenogram of the chest about a year after the onset of her illness showed many nodular shadows in the lower half of each lung field and a pleural reaction at the bases (Fig. 2*a*). Several weeks after roentgen therapy to the chest, these shadows all disappeared (Fig. 2*b*). The lesions were thought to be due to leukemic thromboses of pulmonary vessels or possibly interstitial infiltrations. (See Fig. 2*c*, photomicrograph from a patient with a similar history.)

Comment. Thromboses due to leukemic cells occur so frequently in this disease that their appearance in pulmonary vessels is to be expected. The roentgenologist should include such a possibility in his interpretation when nodular shadows are seen in the lungs.

Carcinoma.

CASE III. V. F., male, white, aged thirty-nine,

archeologist known to have had several tropical diseases. Complained of chronic cough, increasing dyspnea, weakness and anemia for four years. All studies were negative except for sputum repeatedly positive for *Monilia*. Serial roentgenograms showed diffuse linear and nodular shadows in the lung, becoming progressively confluent until they resembled a "snow storm" just before death (Fig. 3, *a* and *b*). Because of the duration of the illness, the process was considered an infection, possibly moniliasis. Postmortem examination showed carcinoma of the bile ducts with emboli in the pulmonary vessels (Fig. 3*c*). The tumor was peculiar in that it produced marked connective tissue reaction which "choked" it by dense scarring and accounted for its protracted course.

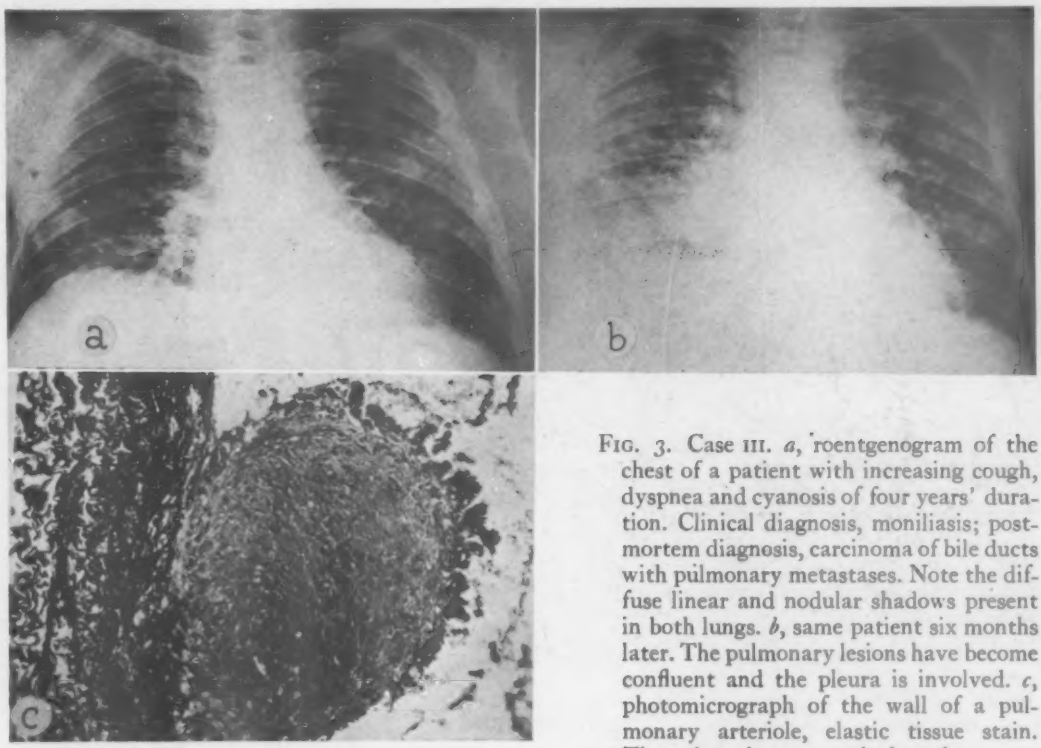

Fig. 3. Case III. *a*, roentgenogram of the chest of a patient with increasing cough, dyspnea and cyanosis of four years' duration. Clinical diagnosis, moniliasis; postmortem diagnosis, carcinoma of bile ducts with pulmonary metastases. Note the diffuse linear and nodular shadows present in both lungs. *b*, same patient six months later. The pulmonary lesions have become confluent and the pleura is involved. *c*, photomicrograph of the wall of a pulmonary arteriole, elastic tissue stain. There is a large mural thrombus composed of connective tissue with a rim of degenerating tumor cells. This illustrates the hematogenous spread of the tumor, and the restraining effect of the connective tissue reaction to it which accounted for the protracted course of the disease.

Comment. This sequence of events is not rare with tumors of the biliary and gastrointestinal tracts. Occasionally, a patient may present right heart failure, because of the extent of the pulmonary vascular occlusion by metastases, as the first symptom of such a tumor.[6]

3. *Intrinsic Disease of Vessel Walls.*

Acute rheumatic fever.

Case IV. E. S., male, white, aged twenty-five, with known rheumatic heart disease. Acute exacerbation with sore throat, fever, rash, and abdominal pain. Living and well ten years later. Roentgenogram at height of the illness showed a diffuse hazy shadow obscuring the upper half of each lung without evidence of cardiac failure (Fig. 4*a*). After recovery, follow-up roentgenograms have revealed an unchanging pattern of diffuse, symmetrical nodulation, probably representing organization of areas of perivascular exudation, possibly with deposition of hemosiderin (Fig. 4, *b* and *c*).

Comment. This is a good example of the

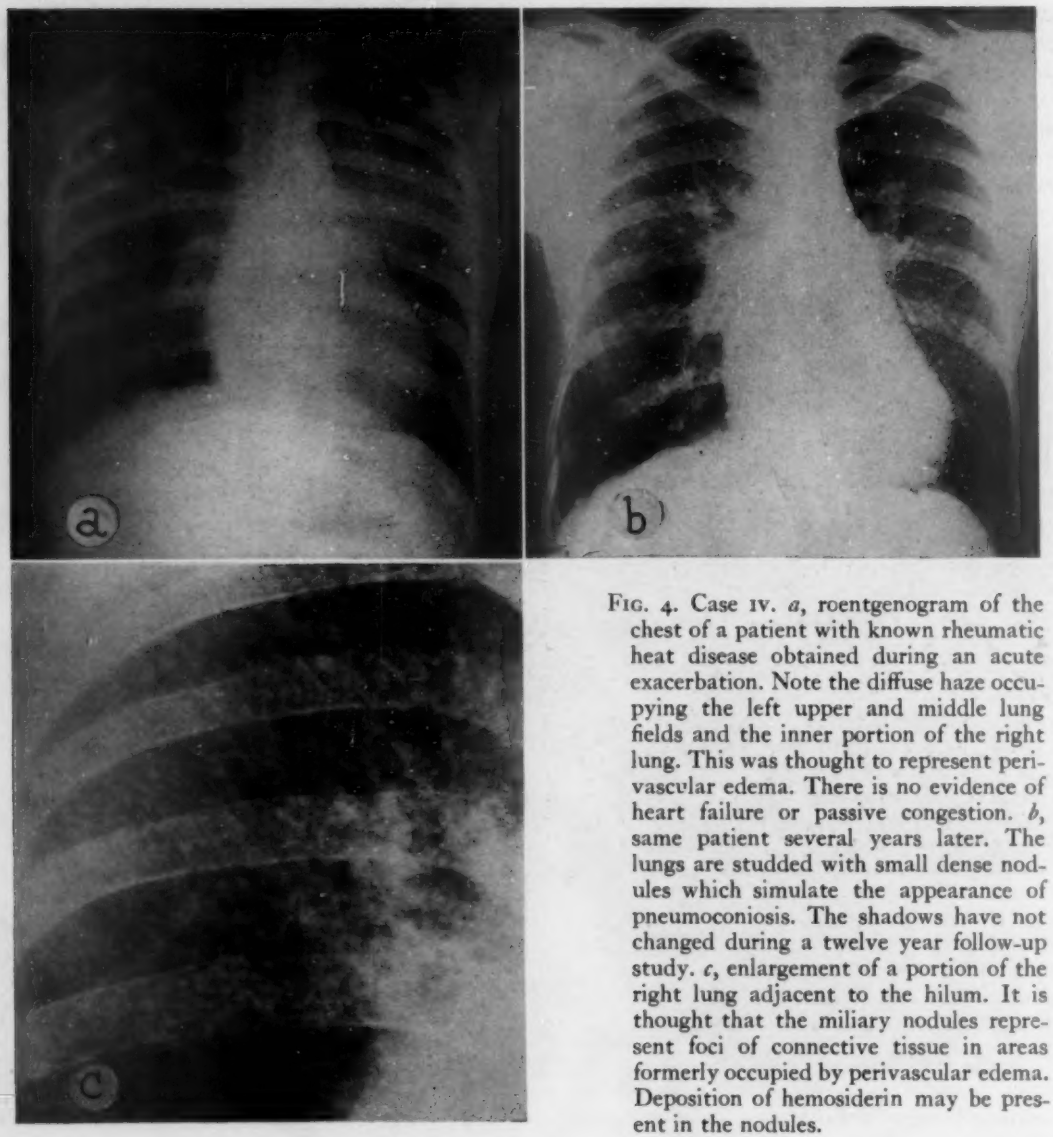

FIG. 4. Case IV. *a*, roentgenogram of the chest of a patient with known rheumatic heat disease obtained during an acute exacerbation. Note the diffuse haze occupying the left upper and middle lung fields and the inner portion of the right lung. This was thought to represent perivascular edema. There is no evidence of heart failure or passive congestion. *b*, same patient several years later. The lungs are studded with small dense nodules which simulate the appearance of pneumoconiosis. The shadows have not changed during a twelve year follow-up study. *c*, enlargement of a portion of the right lung adjacent to the hilum. It is thought that the miliary nodules represent foci of connective tissue in areas formerly occupied by perivascular edema. Deposition of hemosiderin may be present in the nodules.

controversial "rheumatic pneumonia." The acute lesion of rheumatic fever is a periarteritis, accompanied by increased permeability of the vessel wall. It may occur in the lung as elsewhere throughout the body.

4. *Obliteration of Vessels by Adjacent Disease.*

Tuberculosis.

Many acute and chronic pneumonias produce vascular occlusion which hinders natural body defenses and prevents repair.[4] This is best illustrated by a study of the vascular system of excised lungs after injection of the vessels with radiopaque material as Birkelo and Brosius have shown (Fig. 5, *a* and *b*). When attention is centered upon the pulmonary consolidation occurring in most diseases of the lung, the degree of vascular damage is often overlooked.

5. *Hypersensitivity States.*

Periarteritis nodosa.

CASE V. B. S., male, white, aged forty-five,

hospitalized because of rapid loss of 30 lb. in weight, severe pains in all extremities, asthmatic attacks, and asthenia. Found to have a basal metabolic rate of plus 50 per cent and gallstones. Initial diagnosis was hyperthyroidism and peripheral neuritis due to avitaminosis. Developed several attacks of sudden respiratory embarrassment with cyanosis requiring oxygen, in one of which death ensued. Admission roentgenogram, July 15, 1939, showed nothing abnormal in the chest (Fig. 6*a*). During a sudden attack of extreme respiratory distress on August 4, 1939, extensive changes were seen in the inner two-thirds of each lung field compatible with edema due to increased capillary permeability (Fig. 6*b*). No evidence of heart failure was present. Symptoms ceased abruptly after forty-eight hours and the follow-up roentgenogram on August 14, 1939, showed remarkable clearing of the shadows (Fig. 6*c*).

Comment. The pulmonary manifestations of periarteritis nodosa are mentioned infrequently.[7] These patients all have a long history of "asthmatic" attacks which are probably transient episodes of pulmonary edema, progressively severe, and ending in respiratory failure. It is not widely realized that subacute or chronic states resembling periarteritis nodosa may exist, and that a mild form of the disease may occur in epidemic form.[5]

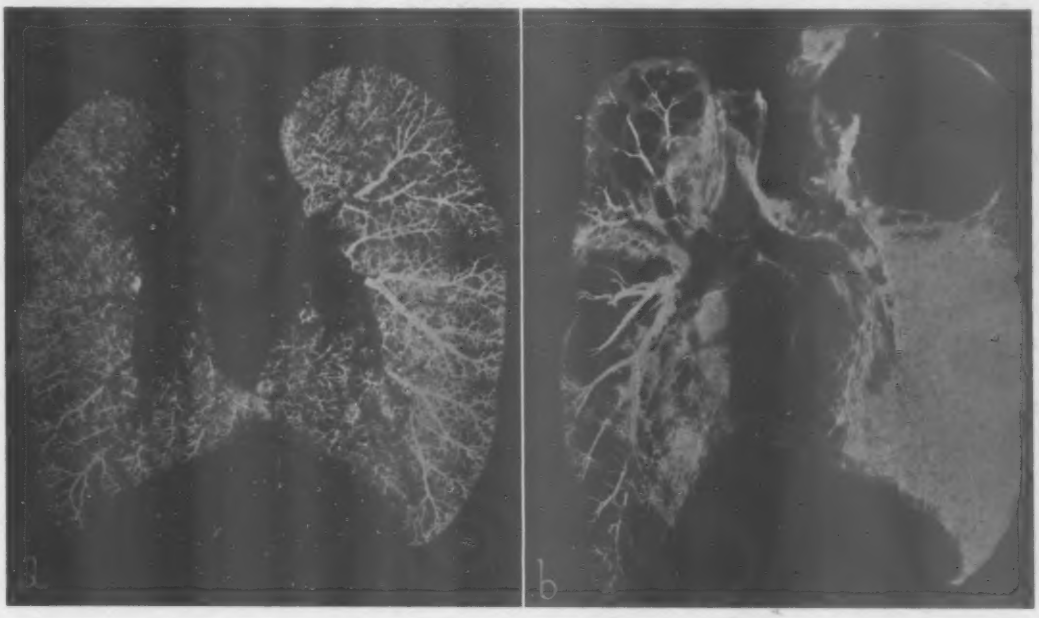

FIG. 5. (Kindness of Drs. Birkelo and Brosius.) *a*, roentgenogram of excised normal lungs after injection of the vascular system with opaque material. Note the lacy pattern and the extensive ramifications of the small vessels at the pleural surfaces. *b*, roentgenogram of excised tuberculous lungs under similar conditions. Note the marked reduction in vascularity of the right lung in particular. It is thought that this degree of impairment of blood supply is an important factor in preventing healing of the disease.

CONCLUSION

Reference to Table 1 will indicate the diversity and extent of the pathology touched upon in this paper. There has been presented an illustration, with explanatory notes, of each of the main headings in the table. By this means it is hoped to advance a new point of view in regard to vascular lesions in the lungs which may be useful in the differential diagnosis of many otherwise obscure clinical conditions.*

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* For discussion see page 37.

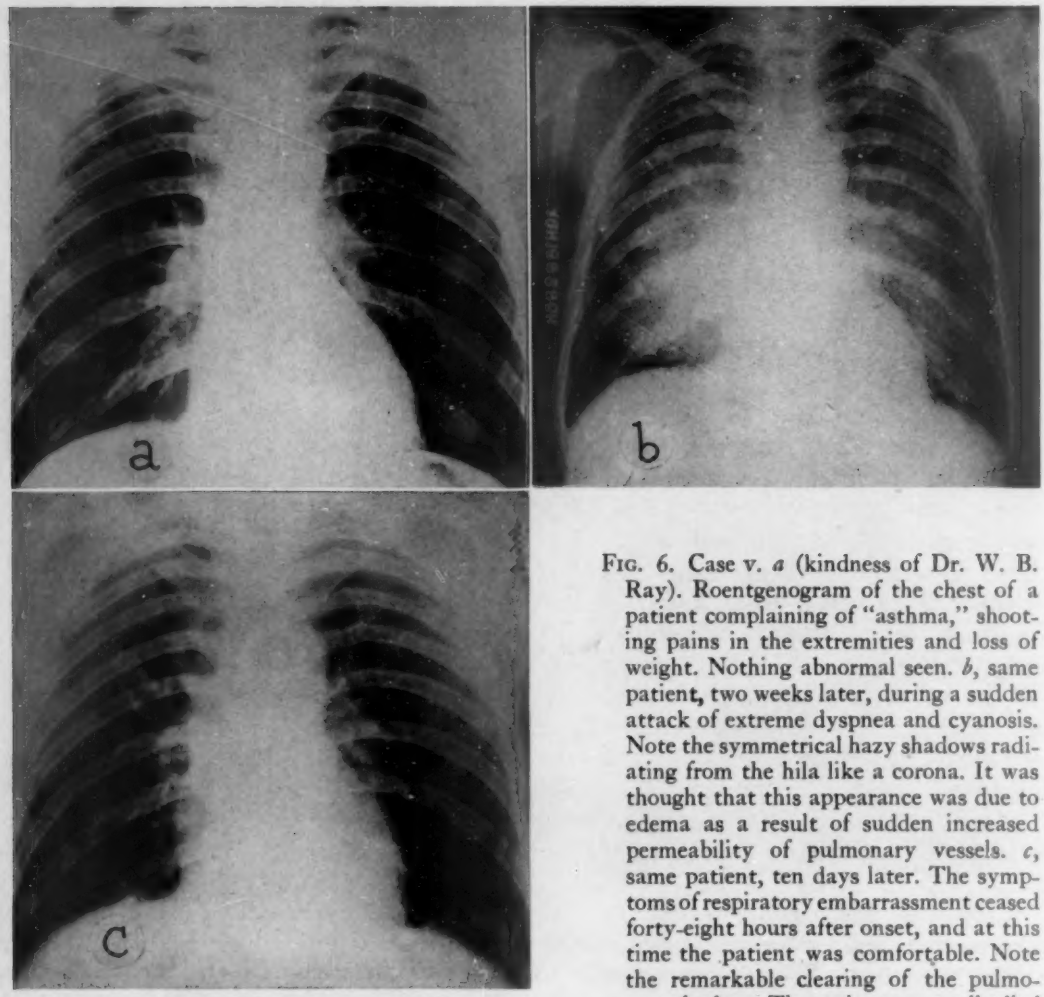

FIG. 6. Case v. *a* (kindness of Dr. W. B. Ray). Roentgenogram of the chest of a patient complaining of "asthma," shooting pains in the extremities and loss of weight. Nothing abnormal seen. *b*, same patient, two weeks later, during a sudden attack of extreme dyspnea and cyanosis. Note the symmetrical hazy shadows radiating from the hila like a corona. It was thought that this appearance was due to edema as a result of sudden increased permeability of pulmonary vessels. *c*, same patient, ten days later. The symptoms of respiratory embarrassment ceased forty-eight hours after onset, and at this time the patient was comfortable. Note the remarkable clearing of the pulmonary shadows. The patient eventually died and postmortem examination showed periarteritis nodosa.

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Vol. 61, No. 1

LUNG FUNCTION*

By ROBERT D. DRIPPS, M.D.
PHILADELPHIA, PENNSYLVANIA

MEDICINE has passed beyond the stage of descriptions of disease entities, of purely anatomic considerations and postmortem reconstructions, of qualitative estimates of function, into the stage of accurate quantitative measurements of the changes which disease imposes on the living body. As part of this symposium on the lesser circulation there follows a brief review of some of the common methods of estimating pulmonary function and of some of the more recent methods which have been developed.

The oxygenation of the blood and the removal of its carbon dioxide are functions of the lungs which normally, even during moderate exercise, are accomplished without the occurrence of dyspnea or discomfort. One of the earliest symptoms of cardiorespiratory dysfunction is the appearance of dyspnea during slight exertion, a symptom which the patient himself notices. The patient, however, cannot evaluate objectively the degree of disability. It is for this reason that various methods have been developed to measure the function of the lungs. These methods may be divided into four main groups:

1. Static measurements of the volume of the lungs include the determination of vital capacity, total lung volume, and functional residual air.

Vital capacity is determined in the ordinary manner. Residual air is that volume of air remaining in the lungs after a maximum expiration. In man residual air is measured by indirect methods such as the dilution technique, in which the subject breathes 100 per cent oxygen in order to eliminate most of the nitrogen from the lungs. If the initial proportion of nitrogen in the lungs is known and the volume of nitrogen eliminated is measured, the total lung volume can be calculated. By subtracting the vital capacity from the total lung volume the volume of residual air is obtained.

These relatively simple methods can supply important information regarding pulmonary abnormalities. For example, in emphysema the (functional) residual air is increased to almost one-half of the total lung volume. Such a proportion of residual air results in a very definite change in respiratory function due to inadequate mixing of the inspired air.

There has recently been developed an apparatus for the continuous determination of the nitrogen content of a gas mixture. This instrument contains an evacuated tube into which is sucked the gas to be studied and through which is passed a 2,000 volt current. In the presence of nitrogen a characteristic color is produced which is more intense the more nitrogen is present. The principle is similar to that of ordinary neon signs. Using this device to analyze continuously the nitrogen content of the expired air, one is able to obtain within a few seconds data which formerly were acquired much more laboriously. It is thus now possible to determine in large groups of individuals of all ages the normal volumes of dead space, tidal air, residual air, and total lung volume, and to test similar large groups of individuals having impaired respiratory function.

2. The second group of methods might well be called dynamic ventilation tests. The rate, the depth, the minute volume of respiration, and other factors can be measured in relation to rest and exercise.

The maximum breathing capacity can be measured by having the subject breathe into a spirometer for thirty seconds as rapidly and deeply as possible. Some physi-

* From the Division of Anesthesiology, Hospital University of Pennsylvania and the Harrison Department of Surgical Research, University of Pennsylvania School of Medicine. Presented at the Forty-eighth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 16–19, 1947.

cally fit individuals can breathe as much as 175 liters of air per minute. If the normal maximum breathing capacity is known for an individual, a subsequent reduction of this capacity signifies a diminished pulmonary reserve.

The velocity of air flow in the trachea can be measured by exposing to the air flow a wire so fine that it will be deflected by the velocities usually present. The amount of deflection can be measured and recorded. Another instrument for measuring such velocities consists of a fine wire mesh with manometers on either side. The passage of air through this instrument sets up a differential pressure gradient which increases with the velocity of the flow. Measurements of this sort are essential in the development of equipment for oxygen therapy. It is not generally recognized that a dyspneic individual whose minute volume of respiration is 20 liters must at the peak of inspiration move air at the rate of 60 liters a minute. If oxygen cannot be delivered at this high rate the patient will be inadequately supplied with oxygen for a short period during every inspiration. Since such velocity determinations give some indication of an individual's ability to expel material from the respiratory tact, they are also of interest in patients with atelectasis, pneumonia and fibrosis.

In a certain proportion of normal subjects, the inspired air will be unequally distributed between the various lobes of the lung with the result that the oxygen saturation of the blood leaving a lobe will vary depending upon the degree of efficiency of ventilation in that lobe. Such inequality in the distribution of inspired air is more often found in patients with pulmonary disease in which secretions or other obstructions may bar access to some alveoli. The degree of mixing in the lung can be measured by determining the amount of nitrogen in a sample of the alveolar air of a patient who has breathed 100 per cent oxygen for seven minutes. Normally at the end of seven minutes of breathing pure oxygen the nitrogen will be less than 2 per cent of the alveolar air. In patients who have unequal lung mixing due to emphysema the sample of alveolar air may contain 5 or 7 per cent of nitrogen.

3. Another group of measurements are concerned with the diffusion of gases between the alveoli and the blood. The arterial blood can be analyzed for its pH and for its oxygen and carbon dioxide content. From such data the tension or pressure of the gases in the blood leaving the lungs can be calculated. Direct measurements of the tension also can be made but the technique is complicated and time consuming.

Since it is the pressure of the oxygen in the plasma which drives the oxygen molecules into the tissue cells, a knowledge of the tension of oxygen in the arterial blood is of more importance in determining the degree of anoxemia than a knowledge of the percentage oxygen saturation of the hemoglobin. Yet clinicians are usually content to estimate the degree of anoxemia by the oxygen saturation of the hemoglobin or by the cyanosis which the patient may exhibit. Unfortunately, cyanosis is rarely apparent until the oxygen in the hemoglobin has fallen from a normal value of 97 per cent to 85 or 80 per cent (at which point the tension of oxygen in the blood may have fallen from the normal of 100 mm. of mercury to 70 mm.), and a considerable reduction of the tension of oxygen can occur before any perceptible decrease in oxygen saturation can be detected by chemical examination. This latter fact is made possible by the peculiar ability of hemoglobin to retain oxygen, as illustrated by the shape of the oxygen dissociation curve of hemoglobin. Therefore, although a decrease in the saturation of hemoglobin with oxygen indicates that the diffusion of oxyen in the lung is inadequate, the tension of the oxygen in the arterial blood is a better guide because it is more sensitive and is altered sooner in disease.

It should be noted that a normal tension of oxygen and of carbon dioxide can be present in the blood even though a lung may have suffered considerable anatomic

damage. For example, if a disease process, such as a large lung cyst, has shut off the blood supply to the affected area of the lung, leaving the remainder of the lung normal, the blood passing through the lung will not be subjected to abnormalities of diffusion and therefore studies of the gases of the blood will be normal, although roentgenologic examination would reveal the abnormality.

4. The last group of measurements are more concerned with pulmonary blood flow. They include measurements of pulmonary circulation time, of the pressure in the right ventricle and in the pulmonary artery, of the volume of the pulmonary blood, and of the cardiac output.

The speed of the pulmonary circulation can be determined by a relatively new device, the oximeter, which when placed on a patient's ear will record the degree of saturation of the hemoglobin by means of a photoelectric cell. As the hemoglobin becomes increasingly saturated and therefore redder in color, more light is transmitted to the photoelectric cell thereby increasing its output of electrical current. If 100 per cent oxygen is breathed, the oximeter will, by increasing its electrical output, show a distinct increase in the saturation of the hemoglobin within five to seven seconds. This is the shortest time in which the excessively oxygenated blood can get from the lungs to the ear. Any delay in this response of the oximeter indicates either a slow circulation through the lungs or the presence of rather uniform and widespread pulmonary lesions such as bilateral pulmonary edema, emphysema, or fibrosis.

Since the techniques of catheterization of the heart are discussed elsewhere in this symposium, that phase of lung function will not be taken up here.

The methods already outlined, dealing with the lungs as a whole, do not distinguish between the function of the right and left lungs. Some of these tests can be performed upon the two lungs separately by the technique of bronchospirometry or catheterization of the individual lungs. Since in these circumstances the breathing is markedly altered, the data obtained cannot with assurance be analyzed quantitatively.

The methods outlined here have been developed through the cooperation of the physicists and chemists who designed the instruments, the physiologists who specified what the instruments should measure and the clinicians whose task will be to apply these new methods to the study of disease. The ability to make accurate measurements easily should prove to be a stimulus to the study of pulmonary function.*

3400 Spruce St.,
Philadelphia 4, Pa.

* For discussion see page 37.

CONDITIONS WHICH RESULT IN INCREASED PRESSURE WITHIN THE LESSER CIRCULATION*

By C. ALLEN GOOD, M.D.
Section on Roentgenology, Mayo Clinic
and
THOMAS J. DRY, M.D.
Section on Cardiology, Mayo Clinic
ROCHESTER, MINNESOTA

HYPERTENSION within the lesser circulation may arise under a variety of circumstances. Until the recent development of the procedure for catheterization of the heart, there was no practical method for measuring the pressure in the pulmonary circuit. Recognition of pulmonary hypertension depended, therefore, on a knowledge of the various mechanisms capable of producing it and on the recognition of the secondary phenomena resulting from its presence. Although new knowledge concerning pulmonary hypertension undoubtedly will be disclosed by catheterization, it is well to review the information which can be gained without resorting to this procedure.

Any mechanism which leads to increased pressure in the lesser circulation increases the work of the right ventricle. If this increased pressure persists, hypertrophy of the right ventricle results. Arteriosclerotic changes in the pulmonary arteries follow and over a period of time, when the reserves are exhausted, failure of the right side of the heart ensues. In certain conditions in which rapid development of pulmonary hypertension occurs, dilatation rather than hypertrophy of the right ventricle takes place. This change is reversible if the cause of the increased pressure is eliminated.

The signs of pulmonary hypertension which are disclosed to the roentgenologist are (1) enlargement of the pulmonary arteries and of the outflow tract of the right ventricle (conus) and (2) enlargement of the right side of the heart. If incompetence of the pulmonary valve is added because of dilatation of the right ventricle, the phenomenon of "hilar dance," an exaggerated pulsation of the pulmonary arteries in the hilus of the lung, also may be seen.

The sign which is present earliest is enlargement of the pulmonary artery and of the outflow tract of the right ventricle, the pulmonary conus. Usually the enlarged pulmonary conus can be seen as a convexity of the upper portion of the left border of the cardiac silhouette. In the right anterior oblique position this convexity is more marked and this portion of the heart shadow may bulge into the retrosternal space.

In addition to enlargement of the conus the branches of the pulmonary artery in the hilus of the lung may appear dilated. Schwedel stated that the descending branch of the right pulmonary artery normally measures between 9 and 14 mm. in diameter. These figures apply only if teleroentgenograms are employed and the artery is measured in its upper portion perpendicular to the bronchus which defines its medial border.

Enlargement of the right ventricle usually appears on the posteroanterior roentgenogram of the thorax as enlargement of the heart shadow to the left. The diaphragmatic segment of the heart is found, in the left anterior oblique position, to be increased in size and its ventral border is rounded. Enlargement of the left ventricle will, in this position, produce a rounding of

* Presented at the Forty-eighth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 16–19, 1947.

the dorsal border of the cardiac shadow. As enlargement progresses the tricuspid valve may become incompetent and the right auricle may become dilated; this produces enlargement of the heart shadow to the right.

In a typical case the roentgenologist will see a convex left border of the heart possibly with some enlargement of the cardiac silhouette. The aortic knob may be less prominent than usual because of rotation of the heart on its vertical axis, and the hilar vessels may be dilated.

CAUSES OF PULMONARY HYPERTENSION

The various conditions which cause pulmonary hypertension may be classified into the following groups:[5] (1) obstruction to the lesser circulation beyond the pulmonary circuit, (2) obstruction within the pulmonary system, (3) abnormal shunts of blood from the arterial side into the pulmonary circulation and (4) kyphoscoliosis.

Obstruction beyond the Pulmonary System. The two most common causes of pulmonary hypertension due to obstruction beyond the pulmonary system are mitral stenosis and a failing left ventricle for any cause. In either instance the vessels of the lesser circulation are overloaded and the efferent blood stream from the lungs is impeded. In some cases of mitral stenosis arteriosclerotic changes develop in the arterioles of the lesser circulation,[8] which further adds to the burden placed on the right side of the heart.

In addition to the enlargement of the conus, as seen on the roentgenogram, mitral stenosis produces enlargement of the left auricle. This can best be demonstrated roentgenologically by the posterior displacement of the barium-filled esophagus with the patient in the right anterior oblique position.

Obstruction within the Pulmonary System. Within the pulmonary circuit the lesser circulation may be obstructed at different levels. The main pulmonary vessels may be occluded by an embolus or a thrombotic process.[7] The small and medium-sized branches of the pulmonary arterial tree may become occluded in the course of sickle cell anemia,[13] schistosomiasis[3] or extensive metastatic carcinomatosis.[2] The pulmonary arterioles may become sclerotic[6] or they may undergo obliterative changes in association with many pulmonary diseases. Finally, the capillary system may be obstructed in emphysema and in widespread pulmonary fibrosis[10] such as that found in tuberculosis and pneumoconiosis.

Shunts. Abnormal shunts of blood from the arterial side into the lesser circulation are usually congenital but may be acquired. The most common congenital lesions of this type are patent ductus arteriosus, interatrial septal defect and Lutembacher's syndrome (interatrial septal defect and mitral stenosis). Rarely a shunt may be acquired by the gradual rupture of an aortic aneurysm into the pulmonary artery.[4]

Kyphoscoliosis. Kyphoscoliosis probably causes pulmonary hypertension because the thoracic deformity produces areas of atelectasis and of emphysema in the lung. It is a well known fact that most hunchbacks die of heart failure. Bachmann has shown that in 87 out of 154 cases death was due to failure of the right side of the heart.

A condition which produces pulmonary hypertension and one in which an early roentgenologic diagnosis is often extremely important is pulmonary embolism. In many cases of pulmonary embolism the clinical features are so typical that roentgenologic aid is unnecessary. In others, particularly in those in which the clinical features are minimal or misleading, the roentgenologic signs may be of inestimable aid in arriving at a proper diagnosis.

Westermark has stated that the roentgenologic sign of pulmonary embolism unaccompanied by infarction is a decreased density of the segment of lung supplied by the occluded artery. This appearance is due, he has said, to the lack of blood in that portion of the pulmonary arterial tree.

In many instances the roentgenologist may see the signs of acute cor pulmonale[12] (Fig. 1). These are dilated pulmonary ves-

sels in the hilus, enlarged conus shadow and enlarged heart. It is probable that the sudden change in pressure in the lesser circulation is due to generalized spasm of the arterioles of the lung as well as to occlusion of a larger vessel. At any rate, if the patient survives, canalization of the occluded artery takes place and the pulmonary hypertension subsides. Eventually the heart and pulmonary vessels return to normal size.

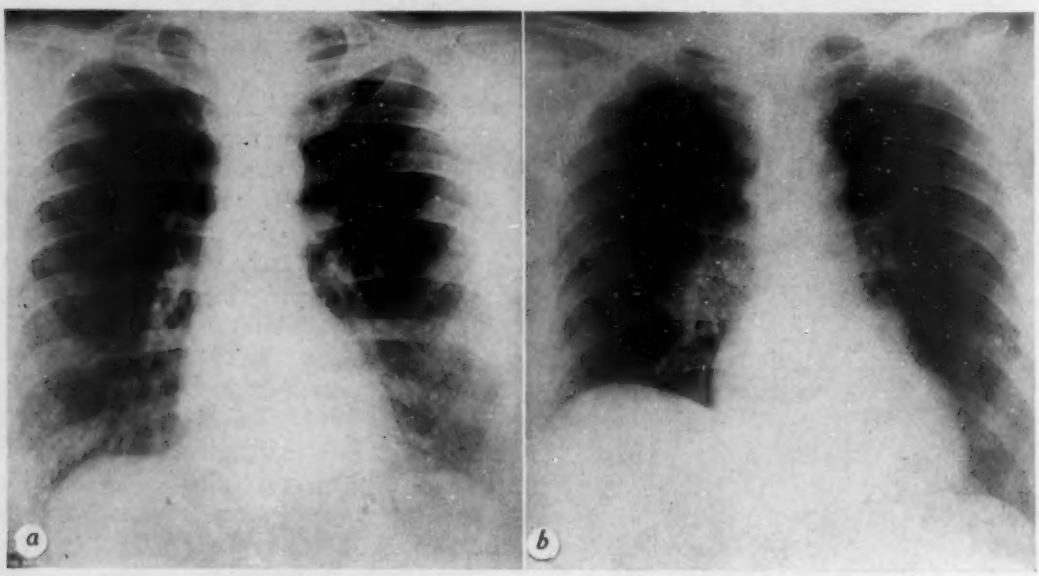

FIG. 1. *a*. Before operation. The size and shape of the heart and pulmonary arteries are normal. *b*. Fourteen days after cholecystectomy and one day after the onset of pain in the right side of the thorax. Signs of acute cor pulmonale are seen in the dilated pulmonary arteries, enlarged heart and convex upper segment of the left border of the cardiac shadow. The patient died a few hours after this roentgenogram was made. An embolus in the right pulmonary artery was found at necropsy.

It is well to point out here that a roentgenologic diagnosis of pulmonary embolism based on the signs of pulmonary hypertension is often possible before the roentgenologic signs of pulmonary infarction and of pleural reaction appear. In such cases therapeutic measures can be instituted earlier, a feature which may save the life of the patient.

SUMMARY AND CONCLUSIONS

Pulmonary hypertension is a secondary manifestation of a wide varitey of conditions which exert their effects either by obstruction of the lesser circulation in or beyond the pulmonary circuit, or by shunts of arterial blood into the pulmonary circulation. Regardless of the mechanism responsible for its production, pulmonary hypertension is recognized by the secondary effects on the cardiorespiratory system eventuating in right ventricular hypertrophy or dilatation, changes characterized roentgenologically by enlargement of the pulmonary artery and conus. Absence of enlargement of the left auricle is evidence that the obstruction is within the pulmonary circuit.

Early diagnosis of pulmonary embolism, especially in those cases in which minimal or misleading clinical features occur, is possible by the recognition of the roentgenologic signs of acute cor pulmonale.*

Mayo Clinic
Rochester, Minn.

* For discussion see page 37.

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THE EVALUATION OF THE LESSER CIRCULATION AS PORTRAYED BY THE ROENTGENOGRAM

By W. WALTER WASSON, M.D.
DENVER, COLORADO

THE lesser circulation is one of the last of the major systems to yield to exploration and evaluation by means of the roentgen examination. The lesser circulation is unique in that it is completely enclosed within the thorax and therefore is very difficult to explore by direct observation. There has been considerable controversy as to who is entitled to the credit for the first description of the lesser circulation. Some historians contend that Servetus, Realdus Columbus or Ibn Nafis was each the discoverer of the lesser circulation. However that may be, very little was contributed to our knowledge of the lesser circulation until the latter part of the Nineteenth Century. Until the last few years, the investigation of the lesser circulation has been done by physiologists and by animal experimentation. These researches were very ingenious and were carried on by many investigators in the various fields of anatomy and experimental physiology. It is most interesting to find that the present day direct investigation of the lesser circulation has proved the accuracy of the physiologist.

The final evaluation of the lesser circulation by means of the roentgen examination is dependent upon the correct portrayal of the anatomy of the chest, its physiological and mechanical variations and the various disease conditions which may be present. The portrayal of the anatomy of the lungs and heart is dependent upon the air content of the lungs. Without air in the lungs, there is no differentiation of the anatomical structures. When the lungs are well inflated, the anatomical structures are visualized, both as to size and position and character, by the displacement of the air content. The pathologic conditions must disturb the normal position or relationship of the air within the thorax in order to reveal their presence and character. Therefore, full inspiration is very essential in the making of the roentgenogram of the chest. This is not always easy, as frequently inspiration is not complete and may be confusing in the study of the air content in the lungs (Fig. 1). Again, expiration may be utilized to demonstrate the presence of emphysema as noted by Westermark.[1] The total lung capacity is twenty times that necessary for aeration of the blood during periods of rest.[2] Also, there is a definite relationship between the air content of the lungs and the blood volume.[3] Without motion, there is no lymphatic flow within the lungs, and it is necessary that any free fluid, either blood or lymph, be outside the blood or lymph vessels and in the air sacs or bronchi where it will interfere with the presence of the air content in order that it may be demonstrated by the roentgen ray. Hemorrhage or lymph stasis sufficient to involve the interstitial framework of the lung also involves the air sacs. Aneurysms of the arteries or veins may be demonstrated by their size.

The technical factors in the making of the roentgenogram of the chest are quite important. This is particularly true in the study of the lesser circulation where the highest quality of definition and contrast is necessary. If the film is not exposed in one-twentieth of a second or less, motion of the heart and vessels will interfere with the sharpness of the delineation on the film.[4] Likewise, underexposures or overexposures

[1] Westermark, N. On bronchostenosis; a roentgenological study. *Acta radiol.*, 1938, *19*, 285–336.

[2] Wasson, W. W. Intrapulmonary air pressure and its relation to pulmonary capillary flow. AM. J. ROENTGENOL. & RAD. THERAPY, 1946, *55*, 575–587.

[3] Heger and Spehl. Quoted by Coryllos, P. N., and Birnbaum, G. L. The circulation in the compressed, atelectatic and pneumonic lung. *Arch. Surg.*, 1929, *19*, 1346–1424.

[4] Wasson, W. W. Radiography of the infant chest with special reference to the "progression of the chest and the determination of the normal." *Radiology*, 1925, *5*, 365–398.

* Presented at the Forty-eighth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 16–19, 1947.

may reveal too much or too little of the anatomy of the lungs. No one position is adequate for the portrayal of the various anatomical structures, but, rather, many different positions are necessary, dependent upon the disease condition to be studied. The required position will tax the ingenuity of the roentgenologist.

A detailed knowledge of the mechanical and physiological variations of the lungs and heart is most essential for the study of the lesser circulation. The infant is born with a certain anatomy, but physiological changes take place very rapidly in the blood flow through the pulmonary vessels,[5] the air content of the lungs, and the relative connective tissue content of the lungs. This connective tissue content of the lungs changes according to the various age periods of life and is influenced by many disease conditions. Such knowledge of the connective tissue content of the lungs at various age periods of life should be had so that the roentgenologist may accurately discuss the presence or absence of pulmonary fibrosis—a most difficult problem in the evaluation of the lesser circulation. Such a study will often raise the question as to when in the human life cycle disease actually begins. This may be very important to the patient with beginning pulmonary fibrosis which will culminate in cor pulmonale. If the roentgenologist is to be of the greatest value to the clinician then, he must be able to foresee that certain conditions of the lungs and heart, as portrayed by the roentgenogram, may lead to serious complications. Serial examinations over a period of months and years not only yield much general scientific information as to the beginning and progress of disease,[4] but will often be of the greatest value to the patient. The heart volume is not a constant factor but will vary according to the general health of the patient as well as to the blood volume and its pressure.

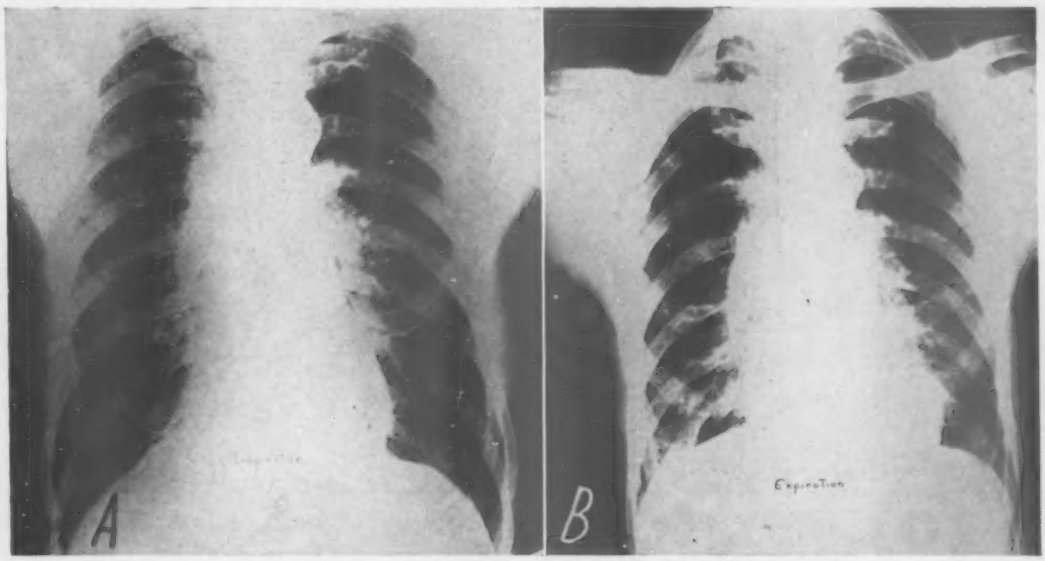

Fig. 1. *A*, complete inspiration; *B*, incomplete inspiration. Illustrating different phases of inspiration which may simulate increased bronchial and arterial densities resembling increased fibrosis.

It has been stated that a demonstration of enlargement of the right ventricle is necessary for the diagnosis of increased

[5] Wasson, W. W. Roentgenographic study of the infant chest as seen at birth. *J.A.M.A.*, 1924, *83*, 1240–1242.

tension within the lesser circulation.[6] Unfortunately, such dilatation of the right heart is a late stage in any disease affecting the lesser circulation, and the goal of the roentgenologist and clinician should be to foresee the possibility of such a dilatation and, wherever possible, to prevent its occurrence. As to whether a dilatation of the right heart will or will not occur is dependent upon whether or not the balance of the mechanical and physiological factors which control the flow of blood through the lungs is sufficiently disturbed. If the clinician or roentgenologist is to have an appreciation of the delicate balance which exists between the flow of blood within the capillaries, the lymphatic flow within the lymph vessels and the to-and-fro movement of the air columns within the bronchi and air sacs, he must be familiar with the circulatory changes which take place at birth, together with the pressure gradient within the pulmonary vessels and the intrapulmonary air pressures (Fig. 2). No satisfactory explanation has yet been presented as to why the blood pressure within the pulmonary vessels drops so rapidly after the first inspiration and maintains such a low pressure as one-fifth to one-sixth that of the greater circulation throughout life. Unquestionably, the intrapulmonary air pressures which change with each inspiration and expiration influence the flow of blood not only through the great veins, but likewise through the pulmonary capillaries[2] (Fig. 3). The pulmonary capillaries lying naked to the air columns within the air sacs[7] must be very sensitive to the changing air pressures. Under normal living conditions, the thickness of the right ventricular wall does not increase after birth, while the thickness of the left ventricular wall increases to two or three times the thickness of the right ventricular wall[2] (Fig. 4). It would therefore seem that there must be another great force other than the propelling power of the right heart which aids in the movement of the great volume of blood through the lungs. Consequently, the thickness of the right ventricular wall and its ratio to the left ventricular wall may be used to calculate the probable pressure in

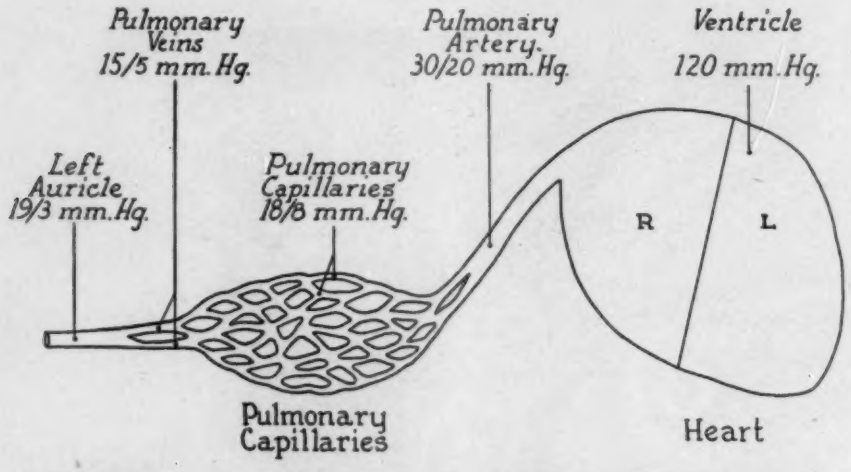

FIG. 2. Diagrammatic representation of the pressure gradient in the lesser circulation.

[6] Schwedel, J. B. Clinical Roentgenology of the Heart. Ann. Roentgenol. Vol. 18, Paul B. Hoeber, New York, 1946.

[7] Maximow, A. A. A Textbook of Histology. W. B. Saunders Co., Philadelphia, 1930.

the lesser circulation if the blood pressure in the greater circulation is known.[2] The demonstration of an enlargement of the right heart by means of the roentgenogram is necessary for a definite statement that there is increased pressure within the lesser circulation, as so many times there appear to be pathological changes within the chest which should increase pressures within the lesser circulation, and yet no such increase exists. Such a demonstration by the roentgenogram is often not easy. With enlargement of the right side of the heart the heart may rotate so that in the posteroanterior view, the heart borders may appear quite normal (Fig. 5). On the other hand, lateral and oblique views of the heart may reveal definite enlargement of the right ventricle, the right auricle and the pulmonary conus and artery.

Until quite recently, the portrayal upon the roentgen film of the heart and the lesser circulation depended upon direct visualization of these structures upon the roentgen film and by a contrast medium in the bronchi or the esophagus. As a basis for his evaluation of the lesser circulation as portrayed upon such roentgen films, the roentgenologist had access to the research of many anatomists, physiologists and surgeons. There was one report of the study of the blood pressures within the pulmonary arteries as found at the time of

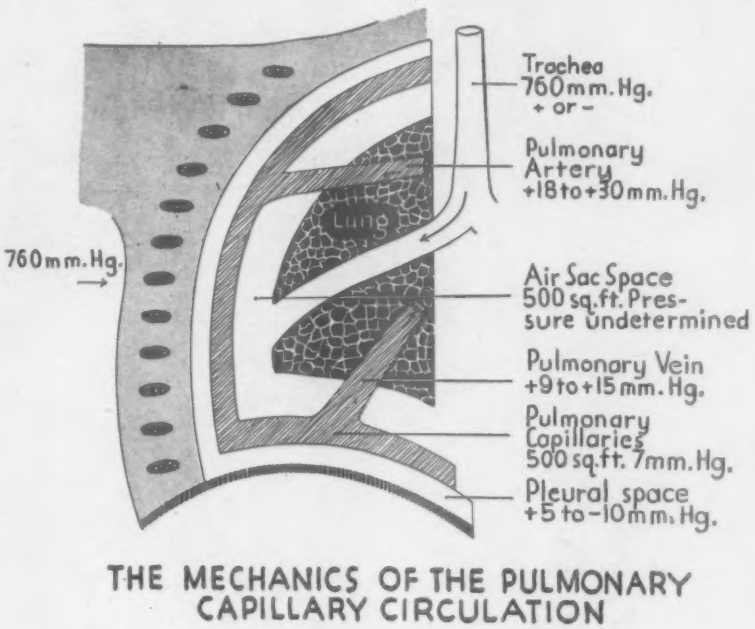

Fig. 3. Sketch illustrating intrapulmonary air pressures, the pulmonary artery, capillary and venous pressures, and the pumping action of inspiration and expiration.

THICKNESS OF VENTRICULAR WALLS AT VARIOUS AGE PERIODS

Age	Left	Right
Birth	*4-4.5 mm.** *0.3 cm.***	*3-4 mm.** *0.3-0.5 cm.***
1 year	*2 X right**	
Puberty	*3 X right**	
Adult	*1.5-2 cm.*** *10-15 mm.****	*0.5-0.7 cm.*** *5 mm.****

* Abt's Pediatrics, 1924
** Pathological Technique, F. B. Mallory, 1938
*** A Text-book of Pathology, Wm. Boyd, 1938

Fig. 4. A chart showing the relative thickness of the right and left ventricular walls at various ages.

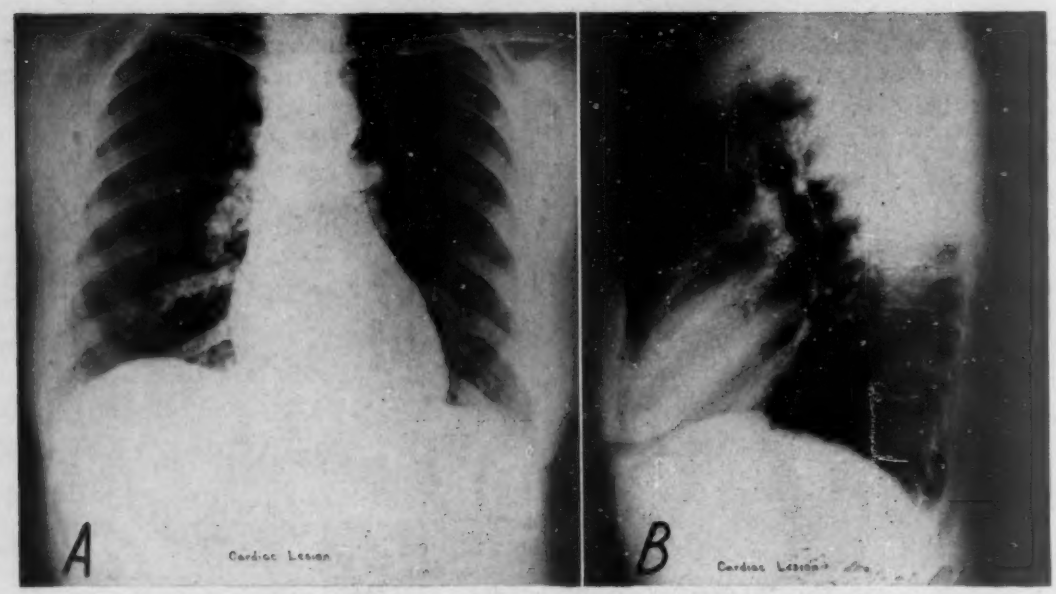

FIG. 5. *A*, heart with normal appearing contours. *B*, same patient, lateral view, full inspiration with diaphragms well down, no kyphosis, but with the lower trachea and bronchi pushed posteriorly by a rotation of the heart with increased diameters; therefore, no shortening of the superior-inferior diameter. A shortening of the superior-inferior diameter from any cause can also produce this appearance.

operation.[8] The determination was made by means of needle puncture. Forssmann's catheterization of the right side of the heart[9] was utilized and elaborated by Cournand and his co-workers[10] who studied the circulation by inserting an x-ray catheter through the veins of the arm and chest and into the right auricle. Their studies stimulated similar investigations by many others. They yielded most valuable data concerning the blood pressure of the lesser circulation and have laid the basis for the surgical correction of many of the congenital heart conditions. Thorotrast has been used for the study of the heart, the great vessels, the pulmonary arteries and veins, and it has enabled the roentgenologist to differentiate aneurysms of the pulmonary vessels and arteriovenous fistulae from other confusing tumors of the lung. Some most interesting arteriovenous fistulae and aneurysms have been reported.[6,11]

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[i] Lauson, H. D., Bloomfield, R. A., and Cournand, A. Influence of respiration on circulation in man. *Am. J. Med.*, 1946, *1*, 315–336.

[11a] Brenner, O. Pathology of the vessels of the pulmonary circulation. *Arch. Int. Med.*, 1935, *56*, 976–1014.

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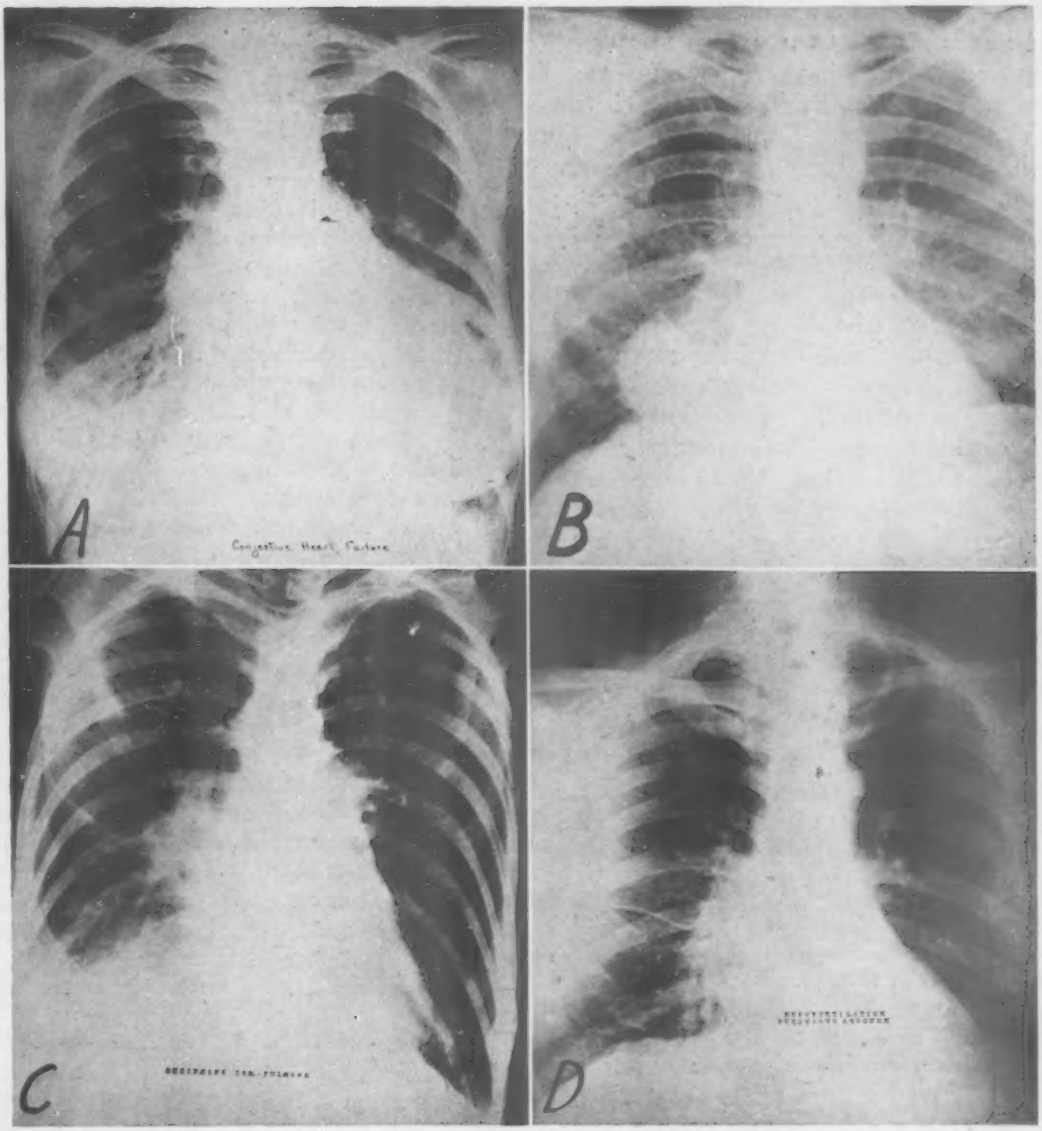

Fig. 6. Four cases illustrating four major classes of disease of the lesser circulation.
A, congestive heart failure; illustrating diseases of the heart, Class 1.
B, arteriovenous fistula (courtesy of Doctors Lawrence and Kerby) illustrating primary disease of the pulmonary vessels, Class 2.
C, cor pulmonale; illustrating diseases of the lungs involving the lesser circulation, Class 3.
D, obesity with hypoventilation; illustrating abnormalities of the thorax influencing the lesser circulation, Class 4.

Another aid in the differential diagnosis is the study of the pulmonary arteries in either lung. Frequently, under pressure the branches of the pulmonary arteries in the lung roots will become dilated and will pulsate to give what has been termed "hilar dance."[6] The pulmonary arteries in the body of either lung may also become prominent and in serial study will give the suggestion of probable increased pressure within the lesser circulation. Finally, there is the lymph stasis or edema of the air sacs

which displaces the air within the air sacs and is readily shown upon the roentgenogram. Such a sign is very characteristic of increased pressure within the lesser circulation and increased permeability of the capillaries or of lymph stasis from any cause. This finding, however, is always associated with atelectasis and many times it is difficult to determine which condition predominates: atelectasis, lymph stasis, or capillary congestion.

A recheck of the list of diseases of the chest and the lesser circulation reveals the fact that there may be a few diseases which are primary to the lesser circulation, but that most of the diseases of the lesser circulation are secondary to other diseases of the chest. In fact, all diseases of the chest may involve the lesser circulation more or less, and even diseases of the greater circulation quite distant from the chest may produce secondary involvement of the lesser circulation. In order to further differentiate and diagnose the diseases of the lesser circulation, it is necessary that some classification be attempted. It seems that there may be four great classes of diseases of the lesser circulation:

1. Disease of the heart, which may be either primary or secondary. It is quite evident that the disease of the heart will be distinctive, clinically or roentgenographically, either as a congenital anomaly or as an involvement in later life. Such disease of the heart may be either primary or secondary and will interfere with the propelling power of the blood through the lesser circulation, as in the congenital anomalies, valvular disease or congestive heart failure, and so forth (Fig. 6*A*).

2. There are a few diseases of the pulmonary arteries and veins which are primary, and which are quite characteristic as a class when viewed roentgenographically: primary sclerosis of the arteries, aneurysms, arteriovenous fistulae and rheumatism. There are also involvements which are secondary to disease elsewhere in the body and which are also characteristic as a class, such as emboli, metastatic tumors, allergic response, and so forth (Fig. 6*B*).

3. There is that group of diseases of the lungs which may involve the lesser circulation secondarily by either interfering with the passage of blood through the pulmonary vessels by pressure or constricting these vessels, as in pulmonary fibrosis, or by direct inflammatory involvement of these vessels, as in pulmonary tuberculosis. This large group is then characterized by those extensive diseases of the lungs producing pulmonary fibrosis, ulceration, and so forth, and result in increased pressure within the lesser circulation and dilatation of the right heart. This final stage has been termed cor pulmonale. Ayerza's disease is considered as cor pulmonale, by most writers[11a,12] (Fig. 6*C*).

4. Finally, there is that group of diseases of the chest which may indirectly influence the lesser circulation by disturbing especially the mechanics of the chest, such as deformities of the chest, obesity, kyphosis of the thoracic spine, the funnel chest, and other anomalies of the thoracic cage which affect the lesser circulation by displacing the viscera or by interference with the correct ventilation of the lungs. Similarly, obesity as described by Kerr[13] will interfere with the proper diaphragmatic action, and therefore interfere with adequate ventilation of the lungs. Such interference with inspiration and expiration and the loss of this power of propelling the blood will throw increased strain upon the right heart, and many of these patients will have cardiac distress. The postoperative fixation of the diaphragms and the resulting hypoventilation of the lung bases, and again increased strain upon the right heart belong in this general class. Trauma of the chest wall with fixation of the chest will produce similar conditions of hypoventilation and increased strain upon the right heart (Fig. 6*D*).

SUMMARY

This paper has endeavored to present the following points:

1. The necessity of complete aeration of the lungs for portrayal of the anatomy of the lesser circulation.

2. The importance of utilizing the correct technical factors in the portrayal of the lesser circulation by means of the roentgenogram.

3. The necessity of a knowledge of the

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[13] Kerr, W. J. Faulty movements of the diaphragm as a cause of non-obstructive emphysema and angina pectoris. *Radiology*, 1942, *39*, 153–156.

physiology and mechanics of the lesser circulation, and the beginnings of diseases for the evaluation of the right heart and increased pressures within the lesser circulation.

4. Four general patterns of disease of the lesser circulation are presented.

DISCUSSION OF SYMPOSIUM ON THE LESSER CIRCULATION*

Dr. Wendell G. Scott, St. Louis, Mo. Six years ago in December, 1941, Dr. Wasson conducted the first symposium on the lesser circulation at the meeting of the Radiological Society in San Francisco. At the conclusion of that symposium, the discussion was opened by Dr. W. E. Chamberlain, and from the presentations he said, "We have had a fine discussion of the gross anatomy and of the basic problems in pathology and physiology. The problem of the lesser circulation is very similar to that presented by a newborn infant: What is it going to develop into?"

This morning we have certainly seen what has developed in those seven years. Dr. Sante gave an excellent demonstration of the detailed anatomy of the lung. Miller's book had been out for several years, but it remained for Dr. Sante and the workers in that field to correlate those anatomical studies with the clinical findings.

Six years ago who of us would have expected to make a diagnosis of arsenical dermatitis, of lupus erythematosus, of trauma to the blood vessels of the lung, of periarteritis nodosa, or of acute rheumatic pneumonia from the roentgenogram? Dr. Barden in his excellent discussion brought this out to us and emphasized the important point that we must be familiar with the clinical findings in the patient whose films we are studying. In that way we sometimes can indicate to the clinician further studies to make the diagnosis.

Dr. Good gave a fine summary of the usual roentgenographic signs and problems of the lesser circulation. It was interesting to hear that the cause of death in hunchbacks is largely due to cardiac failure. I did not fully realize the tremendous significance that kyphosis and distortions of the thorax might play in influencing the cardiac symptoms.

Dr. Wasson has gone over the clinical problems of the lesser circulation. There is no need to repeat them here. In closing, Dr. Wasson, your infant of 1941 has grown into a Miss America, both in chronological age and as a national symbol, as most of the contributions in the diseases of the lesser circulation have been made by American physicians and American scientists.

Dr. David A. Cooper, Philadelphia, Pa. When a clinician goes to a radiologist, it is usually to gain knowledge—and that is pretty much my position here today. Among all the medical specialists, the radiologist is in the most unique position to observe living pathology and pathological physiology in the lung. I would like to congratulate your Program Committee as well as the various essayists who did beautiful jobs, but particularly the Committee that planned this anatomical, physiological and roentgenological approach to the study of a section of the body and its diseases where there is still something to be learned.

This group of papers has brought out the important manifestations in the lesser circulation of many and varied processes, and has again stressed the importance of considering man as a heart-lung preparation rather than one of independent organs, with diseases limited to each. From a clinical standpoint, our job is to recognize symptoms, and then with the aid, most often of the radiologist and the physiologist, to interpret them and correct them if possible.

The importance of pulmonary manifestations in heart disease or the cardiocirculatory manifestations of pulmonary disease have been stressed this morning; also the manifestations in the pulmonary circulation of various constitutional diseases. Time does not permit, nor does my knowledge justify, any attempted detailed discussion of these beautiful presentations. Taken together, they all give us a clearer picture and a better understanding of the effect in the lesser circulation of various disease states.

There is one thing in connection with Dr. Wasson's comments, and the only thing that has not been mentioned: his reference to the influence of respiration on the maintenance of circulation. It has recently been shown by Thompson, of New York, that after stopping

* Papers by Drs. Sante, Barden and Cooper, Dripps, Good and Dry, and Wasson.

the heart, circulation can be maintained by maintaining respiratory motion.

As an internist I want to thank you all for the privilege of being here and learning from the radiologist.

DR. JESSE E. EDWARDS, Rochester, Minn. As a pathologist, it is a great privilege for me to appear before this Society, and it is a distinct pleasure to take part in the discussion of this symposium, a symposium which obviously has been well planned and well executed.

I shall confine my remarks to the circulation of the bronchial arteries. The lesser circulation and the greater circulation are closely interrelated; and one of the important bridges between these two circulations is the system of bronchial arteries. I shall show several lantern slides, the first three of which were loaned to me by Dr. A. A. Liebow of the Yale University Medical School, Department of Pathology.

(Slide) The first three slides are injection preparations of the bronchial artery and pulmonary circulation, as well as of the bronchial system in human material. The bronchial arteries are in black, as is the aorta. The trachea and the bronchi, as well as the ramifications of the bronchi, are in white. The red portrays the location of the pulmonary arterial system; the green represents the pulmonary veins.

In this preparation, which is that of the normal bronchial circulation, small vessels—the bronchial arteries—arise either directly from the aorta or from the intercostal arteries. In the normal circulation, with the injection technique employed, the arteries are not seen in the pulmonary parenchyma. Dr. Liebow and his colleagues have shown that in the presence of bronchiectasis the bronchial arteries are increased in size.

(Slide) In this slide one can see that in bronchiectasis, anastomosis between the bronchial arteries and pulmonary arteries becomes visible. Here is a bronchus which is dilated, and we find here a bronchial artery; here is a branch of it, and here is a pulmonary artery. At this point the two systems visibly anastomose. There are actually numerous anastomoses in this area.

Recently, we have become interested in study of the lungs in cases of coarctation of the aorta, primarily for the purpose of determining whether or not the bronchial arterial system is altered in this condition. We know that the bronchial arteries arise from vessels which normally take part in the collateral circulation in the presence of coarctation of the aorta. Consequently, perhaps the bronchial arteries, as innocent bystanders, if nothing more, may carry a great volume of blood, and possibly this blood is under greater pressure than is normal.

(Slide) The next slide is a photomicrograph of a section of tissue from the lung in a case of coarctation of the aorta. Here is a cross section of a bronchus, showing the epithelium of that structure. In the lower part of the field is a portion of the bronchial cartilage. You will notice, around the epithelium, a vascular plexus, a collar of blood vessels, which probably represents an increase in the amount of blood flowing through those vessels which are supplied by the bronchial arteries.

Not only do we have evidence that the vessels which arise directly from the bronchial arteries carry a greater volume of blood, or blood that is under greater pressure, but there appears to be an increase in the amount of blood in the pulmonary arterial circulation.

(Slide) This is taken from the periphery of the lung, and we find very prominent wide, thin-walled pulmonary arterioles, so that we have here, by inference, if not more, at the present stage of the study, evidence that the pulmonary arteries in the presence of coarctation of the aorta do receive more blood than they do in the normal state.

Not only do the pulmonary arteries appear to be dilated and thin walled, which is evidence that they have been subjected to this stimulus for some time, but also, we find that there is pulmonary alveolar congestion.

(Slide) This slide is a photomicrograph, made with higher magnification, of a section of tissue from the lung, showing a dilated pulmonary arteriole. We see that the capillaries of the alveolar walls are filled and are tremendously distended.

It might be argued that this pulmonary congestion, which is marked, is the result of an acute cardiac failure, from which a number of patients with coarctation of the aorta die. On the other hand, the changes in the larger vessels are distinctly recognizable as anatomic changes, and they undoubtedly represent, as already stated, changes which have developed over a considerable period.

In addition to the congestion, we frequently find evidence of old hemorrhage into the alveolar spaces. It is not uncommon, in study of

sections of the lung in cases of coarctation of the aorta, to find many foci of alveolar spaces filled with hemosiderin-laden microphages, which are interpreted as being evidence of old alveolar hemorrhage.

(Slide) The next two slides represent sections of tissue taken from a patient who had the tetralogy of Fallot. The patient was a fourteen year old girl in whom the usual pulmonic systolic murmur was absent. The clinicians felt that there was complete atresia of the orifice of the pulmonary artery. Pathologically, the valve of the pulmonary artery was almost completely occluded; it admitted only the finest probe. To all intents and purposes, I think, the pulmonary artery was carrying virtually no blood to the lungs.

We have here a photograph of a posterior view of the thoracic organs in that case. Here is the left lung; here is the right lung; here is the esophagus; here is the trachea; over here is the aorta. From the second aortic intercostal artery on the right side we are able to pick up a large bronchial artery, which was easily cannulated and injected. It proceeded to the main bronchus of the right lung, and before that, it gave off a branch which crossed the esophagus and proceeded to the left lung. Undoubtedly, this bronchial artery was the main source of blood supply to the lungs of this patient who had the tetralogy of Fallot.

The observation that the bronchial arteries may carry a substantial amount of blood to the lungs in the presence of pulmonary stenosis is not original with us. In talking to Dr. Liebow the other day, I found that he has demonstrated the same thing recently, and there are reports of similar observations in the literature.

Dr. Felix G. Fleischner, Boston, Mass. I want to use my time as sparingly as possible. Roentgen kymography has been given a new impulse by the application of the photomultiplier tube, the use of which was first suggested in this country by Chamberlain. We in Boston, Dr. Luisada and I, have been using an arrangement similar to, but more sensitive than that devised by Chamberlain and co-workers. We have made observations on the lesser circulation which we thought might be of interest in connection with this morning's symposium.

(Slide) If the pick-up device with the photoelectric tube is placed across the pulsating border of the heart or a great vessel, it operates like a highly sensitive kymograph. While the classical roentgen kymograph registers directly the pulsating shadow on the film, in this new apparatus pulsating changes of light intensity, proportionate to pulsating movements, are being transformed into a pulsating electric current to be recorded by the galvanometer of a standard electrocardiograph.

Placed over the center of an opaque object, e.g. the hilar shadow, changes in the opacity of this object will be recorded by the apparatus. The same thing can be done by placing the pick-up device in any peripheral portion of the lung field.

(Slide) Just to familiarize you with the appearance of these tracings, I show you this slide. In order to time the various waves, we register simultaneously a phonocardiogram which shows the first and second sound. Here you see four different curves: From the left auricle, the left ventricle (at a high level), from the pulmonary artery, and the aorta. You can see the different shapes and amplitudes of these pulsations. They are all taken with the same amplification and sensitivity of the apparatus.

(Slide) You see here curves taken over the right hilum and the right lower lung field. I will not go into any analysis of the details of the curves, but perhaps you recognize that the peak of this curve, which represents the arrival of the pulse wave at the hilum, occurs about a tenth of a second earlier than that in the periphery of the lung field.

(Slide) For better comparison of the time relationship of several curves we can trace two pulsations simultaneously by a tribeam electrocardiograph. Here the lower curve represents the pulsation of the aortic arch, and the upper that of the pulmonary artery on the left side. You see a difference in the shape of the curves, the more pronounced dicrotic wave and an earlier arrival of the pulse in the pulmonary artery.

(Slide) Here I show you another tracing where we have in the lower curve a record of the right hilar vessels, and in the upper curve, with more magnification, the pulsation of the peripheral lung field.

It was most surprising, among our early observations, to see the pulsation of the periphery due to the small arteries of the pulmonary parenchyma, and this has never, to our knowledge, been seen before the application of this new principle. We believe that many interesting observations will be made in the future by this

method, both in the field of physiological research and in that of clinical application.

DR. SANTE (closing). I have only a word to say with reference to coarctation. It seems rather strange to me that coarctation of the aorta should cause such great enlargement of the bronchial arteries. Of course, the site of origin of the bronchial vessels from the aorta or from the intercostal or intramammary is so variable that perhaps the relationship of their origin might have something to do with it. It at least would certainly have something to do with whether or not the bronchial vessels came off above or below the site of coarctation.

It occurs to me that perhaps the explanation might be relatively simple in this regard. If you have a water pipe conveying water of a certain amount and divide it into two smaller divisions, one of which is larger than the other, depending upon the relative size of the two pipes, the amount of water that will go through the smaller will be less due to resistance. Perhaps the explanation is as simple as this. In other words, if you have an aorta of a certain size, which has been coarctated and reduced in caliber, and then a relatively normal sized bronchial artery, it seems obvious that the normal sized bronchial artery, competing with a coarctated aorta, may attain a greater size in proportion because it will get a greater amount of the fluid in proportion.

The fact that the vessels at the periphery of the lung were also dilated in the case that was shown also seems to bear out this reasoning and suggest it as a possible explanation.

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THE VALUE OF ANGIOCARDIOGRAPHY IN ESTABLISHING THE DIAGNOSIS OF PERICARDITIS WITH EFFUSION*

By ROBERT G. WILLIAMS, M.D., *and* ISRAEL STEINBERG, M.D.
NEW YORK, NEW YORK

THE diagnosis of pericarditis with effusion is often difficult. Even after the clinical history, physical examination, laboratory and roentgenological examinations it may be impossible to differentiate pericardial effusion from an enlarged or dilated heart. Angiocardiography has made it possible to visualize the cardiovascular structures and thereby differentiate dilated heart chambers and hypertrophied cardiac walls.[6,7,8] Our experience with 4 cases of pericarditis with effusion forms the basis of this report, and demonstrates how easily and without harm to the patient angiocardiography establishes the diagnosis.

The symptoms of pericarditis with effusion vary considerably from extreme dyspnea to mild pain. Often no symptoms are present until a tamponade effect is produced. The symptoms of cardiac enlargement also vary greatly and are inconstant. In addition, pericardial effusion may be found in the presence of rheumatic heart disease, hypertensive cardiovascular disease, coronary sclerosis, and in the terminal phase of cardiorenal failure. Pericardial effusion is also found in tuberculosis, pyogenic infections, and malignant disease.[1,10] The physical findings in pericarditis with effusion may be a pericardial friction rub, increase in the area of cardiac dullness, distant heart sounds, increased retromanubrial dullness, shift of cardiac dullness on change in position of the patient, and signs of compression of the left lung posteriorly. These signs are produced only in the typical case, are inconstant, and many of them are also present with cardiac dilatation and hypertrophy. The presence of pleural effusion, pulmonary parenchymal consolidation, or atelectasis further complicates the clinical picture and makes the diagnosis of pericarditis with effusion with or without associated cardiac enlargement difficult. Laboratory tests are usually of no direct help in distinguishing between pericardial effusion and cardiac enlargement. They may aid in establishing the presence of infection, heart or kidney disease, but pericardial effusion as a complication cannot be determined. Although the electrocardiogram shows some changes of diagnostic value, there are no specific findings that are characteristic of pericarditis with effusion.[1,12]

The appearance of the cardiac silhouette on roentgenologic examination is probably the most reliable sign of pericarditis with effusion. Roentgenoscopic examination will yield information regarding the pulsation of the cardiac shadow. In pericardial effusion, the pulsations are definitely diminished or absent, especially in the dependent portion of the cardiac silhouette. The usual active pulsation of the ventricular shadow is decreased or absent, and pulsation of the aortic shadow is relatively increased. However, a dilated or hypertrophied heart may produce similar findings. The change in shape of the cardiac shadow with change in position of the patient can be observed roentgenoscopically in a typical case of pericardial effusion. Roentgenographically, pericardial effusion is recognized by marked generalized enlargement of the cardiac silhouette with obliteration of the normal cardiac contours. In some cases a pear or water-bottle shaped shadow with a small aorta above it is formed. Enlargement of the cardiac silhouette most marked in its transverse diameter, with distention of the posterior inferior recess and foreshortening

* From the Department of Radiology, The New York Hospital—Cornell Medical Center, New York, N. Y.

of the vascular shadow most evident with the patient in the recumbent position, and change in size and shape of the cardiac silhouette within a short interval of time can be recognized. These findings again are variable and marked cardiac enlargement will often present the same appearance.[2,9] It is well established that the heart cannot be seen as a separate shadow within the cardiac silhouette in pericarditis with effusion.[4,9] A chronic pericardial effusion may produce little or no effect on the lesser circulation, and no evidence of pulmonary congestion will be seen roentgenologically. However, pericardial effusion with cardiac enlargement is frequently associated with pulmonary vascular congestion. Roentgen kymography records the pulsations of the cardiac shadow and gives no more information than roentgenoscopy.

FIG. 1. Conventional roentgenogram. Female, aged fifty-six, admitted for treatment of active gastric ulcer. Known hypertension for three years. No cardiac complaints. Blood pressure 155/100, fair heart sounds. Left axis deviations in the electrocardiogram. Enlarged cardiac shadow with diminished pulsations roentgenographically. Note the generalized enlargement of cardiac silhouette with absence of pulmonary congestion.

Pericardial tap with removal of fluid is proof of the existence of pericardial effusion, but this procedure may be difficult, and, as Levine[5] points out, is hazardous. The injection of air into the pericardial sac after removal of fluid to visualize the effusion roentgenographically yields little additional information.

METHOD AND FINDINGS

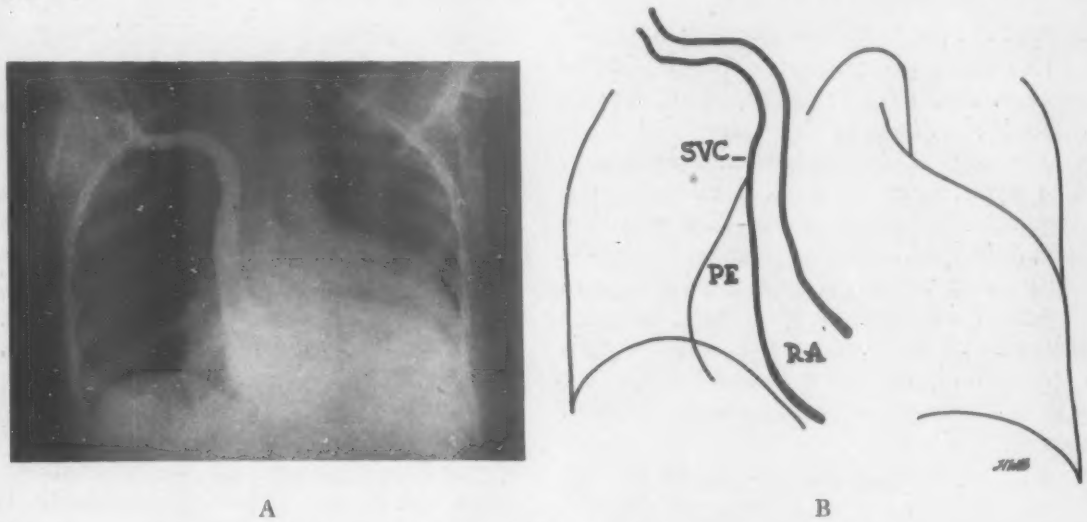
The Robb and Steinberg method of cardiovascular visualization[6,7] was used to study 4 patients in whom routine methods of investigation were inconclusive in establishing the diagnosis of pericarditis with effusion. Films were taken in the posteroanterior projection at appropriate time intervals to visualize the chambers of both sides of the heart. In each instance the opacified right and left cardiac chambers were seen to lie well within the cardiac silhouette, the intervening space representing the chamber walls and the fluid in the pericardial sac entirely surrounding the heart. Pericardial tap with removal of fluid confirmed the diagnosis in each case.

Figures 2 and 3 (Case I) illustrate the angiocardiographic appearance of a pericardial effusion. In this case its presence was not suspected clinically, but was clearly shown by angiocardiography. Case II was a patient who had hypertensive cardiovascular disease and chronic heart failure. He was not suspected of having a pericardial effusion clinically or by routine roentgenographic procedures because the enlarged cardiac silhouette was attributed to the hypertensive disease. Angiocardiographic studies demonstrated the presence of a pericardial effusion in addition to enlargment of the heart chambers. Cases III and IV were two patients in whom angiocardiographic studies revealed the presence of pericardial effusion. Both of these patients had pulmonary tuberculosis, and tuberculosis was believed to be the cause of the pericardial effusion. In one of these cases, repeat angiocardiography following subsidence of the pericardial effusion clearly demonstrated that the opacified cardiac chambers and their walls had resumed their normal border forming position. In all 4 of the patients in this study, no serious reactions were experienced either during or following angiocardiography. None of these patients had severe liver disorders, nephritis, hyperthyroidism,

or known allergic tendencies which are contraindications to the procedure.[6,7]

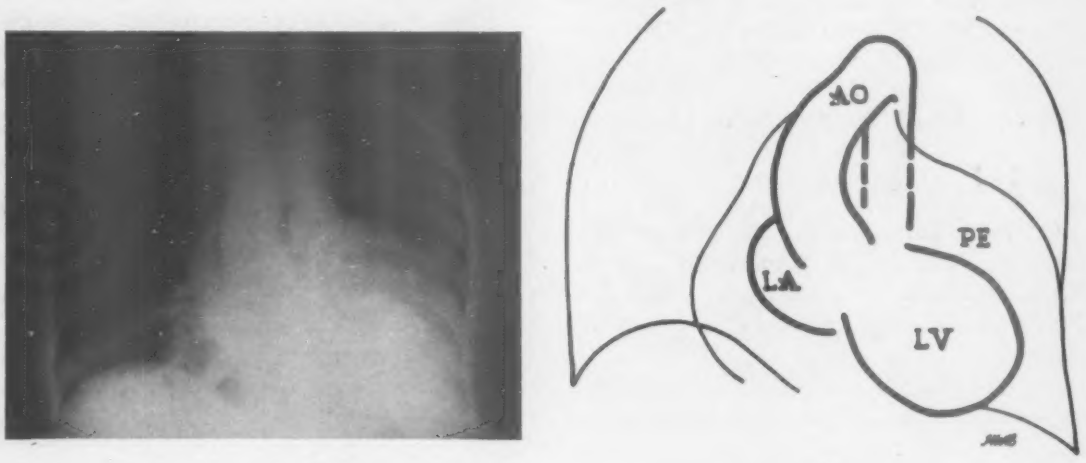
DISCUSSION

Shortly after cardiac visualization was described, it became apparent that this procedure could be used to establish the diagnosis of pericarditis with effusion, particularly in patients in whom clinical and laboratory findings were inconclusive.[6,7] Recently Ungerleider[11] made reference to this possibility. In our 4 cases the presence of pericardial effusion was demonstrated angiocardiographically by the visualization of the opacified cardiac chambers lying within the surrounding shadow of pericardial fluid. Because of the higher concentration of the opaque medium in the right side of the heart, the contrast between the opacified chambers and surrounding peri-



A B

FIG. 2. *A*, contrast roentgenogram at two seconds. Right subclavian, innominate, superior vena cava and right atrium visualized. Note the pericardial fluid shadow adjacent to right atrium. *B*, tracing of *A*. *SVC*, superior vena cava; *RA*, right atrium; *PE* pericardial effusion.



A B

FIG. 3. *A*, contrast roentgenogram at twelve seconds. Pulmonary veins, left atrium, left ventricle and aorta opacified. The shadow of pericardial fluid is seen beyond the left ventricle. *B*, tracing of *A*. *LA*, left atrium; *LV*, left ventricle; *AO*, aorta; *PE*, pericardial effusion.

cardial fluid was more striking than on the left. However, visualization of the left heart chambers in all cases likewise showed the pericardial fluid.

The posteroanterior view was used in these 4 cases, but the same information would be available if patients were studied in other projections. In each instance the patient had no ill effects from the injection of contrast medium. In one case the study was repeated to show the disappearance of the pericardial fluid.

The differentiation between pericardial effusion and cardiac enlargement due to dilated chambers or hypertrophied walls was easily made angiocardiographically. The appearance of cardiac chamber enlargement and thickened cardiac walls has been described in previous reports.[3,8] While the shadow beyond the opacified chambers in pericardial effusion represented both the thickness of the cardiac wall and the pericardial fluid, the diameter of the shadow was well beyond the limits of hypertrophied cardiac walls. Patients with mediastinal masses adjacent to the pericardium were clearly differentiated from pericarditis with effusion by showing the unilateral character of the masses, whereas, in pericardial effusion, both the right and left sides of the heart were surrounded by shadow of the fluid in the pericardial sac. A pericardial cyst has not been encountered, but it is believed that its unilateral location should differentiate it from pericardial effusion.

CONCLUSIONS

(1) Angiocardiography of four patients with pericarditis with effusion demonstrated a characteristic shadow due to the fluid surrounding the opacified cardiac chambers.

(2) The fluid density could be easily differentiated from normal or hypertrophied cardiac walls.

(3) Angiocardiography will establish the diagnosis of pericardial fluid in those cases in which the routine clinical and laboratory findings are inconclusive.

New York Hospital
525 East 68th Street
New York 21, N. Y.

ADDENDUM

Since this paper was submitted for publication, we have demonstrated the presence of pericardial effusion by angiocardiography in 5 additional patients in whom clinical and roentgenologic findings were inconclusive.

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A RAPID METHOD OF ROENTGENOLOGIC EXAMINATION OF THE SMALL INTESTINE*

A PRELIMINARY REPORT

By SYDNEY WEINTRAUB, M.D., *and* ROBERT G. WILLIAMS, M.D.
Department of Radiology, The New York Hospital, Cornell Medical Center
NEW YORK, NEW YORK

AT THE present time there is no standardized technique for the roentgenologic examination of the small intestine, and each roentgenologist has more or less his own method of procedure. Hodges, Rundles and Hanelin[2] for instance, advise one, two and a half, and five hour abdominal films for the initial examination of the small intestine with a second more detailed study when indicated. Schatzki[4] recommends the small intestinal enema first described by Pesquera[3] in 1929. Although this method is rapid, and good visualization of the small intestine is obtained, it necessitates the passage of a duodenal tube, and is practical only in selected cases. Golden[1] examines the entire small intestine by means of roentgenograms taken at half hour intervals supplemented by roentgenoscopy and spot films as indicated. Even though the incidence of small intestinal disease is low, every examination of the small intestine should be thorough and not a casual observation by means of one or two abdominal roentgenograms taken at irregular intervals. The principal disdvantage of any thorough examination is that it requires time-consuming efforts on the part of the patient, the roentgenologist, the technician, and the roentgen department. This is an important consideration when large groups of patients are examined. With all present routine methods of small intestinal examination, the time involved is from three to six hours and frequently longer. With a rapid method of examination, this time factor, and the expense element as well, would be decreased, and there would be more incentive for more frequent investigations of the small intestine.

METHOD AND RESULTS

In 1941, one of us (S. W.) noted the effect produced by an iced drink after a full meal, namely, a marked increase in the motility of the contents of the gastrointestinal tract with resultant diarrhea. Some investigations along this line were conducted by him at that time, which showed roentgenographically that ice water, given after a barium-water meal, produced a marked acceleration of the movement of the meal through the small intestine. We verified these findings in a number of normal cases and found that in about 50 per cent the head of the barium-water meal reached the cecum in one-half hour or less. This procedure was unsatisfactory because there was incomplete filling of the small intestine and distortion of the mucosal pattern. We then conducted small intestinal studies on 90 normal cases using a cold weak solution of sodium bicarbonate. With this method the motility of the gastrointestinal tract was increased above that obtained with cold water alone, but again the mucosal pattern of the small intestine was unsatisfactory. We then decided to use a barium-normal saline meal rather than a barium-water meal. One of the functions of the small intestine is to make hypertonic or hypotonic solutions isotonic. It would be expected that with a barium-isotonic normal saline meal the small intestine would have less work to do, and consequently the transit time would be decreased, and the mucosal pattern would be more normal. Golden[1] employs a barium-saline mixture in gastrointestinal examinations because of its more physiologic action. Accordingly, we determined the comparative motility of a barium-saline meal

* Presented before the New York Roentgen Society, New York, on January 19, 1948, and before the Forty-ninth Annual Meeting, American Roentgen Ray Society, Chicago, Ill., Sept. 14-17, 1948.

TABLE I

PROGRESSION OF BARIUM-WATER MEAL THROUGH SMALL INTESTINE IN ONE HOUR IN 107 NORMAL CASES

Position of Head of Meal	Number of Cases	Per Cent of Cases
Jejunum	32	30
Proximal ileum	19	18
Distal ileum	32	30
Cecum and beyond	24	22

TABLE II

PROGRESSION OF BARIUM-SALINE MEAL THROUGH SMALL INTESTINE IN ONE HOUR IN 114 NORMAL CASES

Position of Head of Meal	Number of Cases	Per Cent of Cases
Jejunum	8	7
Proximal ileum	21	18
Distal ileum	24	21
Cecum and beyond	61	54

and a barium-water meal. First, using a barium-water meal, routine one hour roentgenograms were taken as part of upper gastrointestinal tract examinations. The results are shown in Table I. Note that in 22 per cent of 107 normal cases, the head of the barium-water meal has reached the cecum in one hour. Next, using a barium-normal saline meal, one hour abdominal roentgenograms revealed results as shown in Table II. Note that in 54 per cent of 114 normal cases the head of the barium-normal saline meal has reached the cecum in one hour.

It is apparent, then, that a barium-saline mixture passes through the small intestine more rapidly than does a barium-water mixture. The barium-saline meal also produces less distortion of the mucosal pattern, and more adequate filling of the small intestine.

To utilize the accelerating effect of both saline and of a cold liquid, we devised the following technique of small intestinal examination and present it as a practical and accurate method.

TECHNIQUE OF RAPID METHOD OF SMALL INTESTINAL EXAMINATION

1. Roentgenoscopic and roentgenographic examination of the esophagus, stomach and duodenum (using 4 oz. barium and 4 oz. isotonic normal saline at room temperature).
2. Patient drinks 8 oz. of ice cold normal saline.
3. 14 by 17 inch abdominal film taken at five minutes.*
4. Patient drinks a second 8 oz. of ice cold normal saline immediately after the five minute film.
5. 14 by 17 inch abdominal film taken at fifteen minutes.*
6. 14 by 17 inch abdominal film taken at thirty minutes.*
7. All three abdominal films are shown "wet" to the roentgenologist who roentgenoscopes and takes spot films of suspicious areas, or if the head of the meal has reached the cecum, may roentgenoscope and spot the terminal ileum.
8. If the head of the meal has not reached the cecum, additional films are taken at half hour intervals until it has. The roentgenologist then roentgenoscopes and takes spot films as indicated.

The results of this method in 87 normal and 17 pathological cases are shown in Table III. Note that of 87 normal cases, in

TABLE III

RESULTS OF RAPID METHOD OF SMALL INTESTINAL EXAMINATION IN 87 NORMAL AND 17 PATHOLOGICAL CASES

Head of Barium-Saline Meal in Cecum or Beyond	Normal	Pathological†
½ hour or less	72 (83%)	8
½ to 1 hour	6 (7%)	2
More than 1 hour	9 (10%)	7

† Pathological cases:
2—chronic duodenal ulcer
2—sprue
2—sarcoma second duodenum
5—"deficiency" states
3—active duodenal ulcer
3—regional ileitis
1—ulcerative colitis

* Note that all times are taken from the time of drinking of the first 8 oz. of the ice cold normal saline.

72 (83 per cent) the head of the barium-saline meal had reached the cecum in one-half hour or less. In an additional 6 cases (7 per cent) the meal had reached the cecum in one-half to one hour. Thus, out of 87 normal cases, the head of the barium meal reached the cecum in one hour or less in 78 (90 per cent). In only 9 (10 per cent) did it take more than one hour for the head of the meal to reach the cecum. The mucosal pattern, in all cases, was as good as that obtained on routine small intestinal examinations and in several cases, much better, especially in the terminal ileum where there was a more even distribution of barium. In 42, or about 50 per cent, of the total number of normal cases the barium had entered the cecum in five to fifteen minutes. In several instances the head of the meal had reached the descending colon or rectum at fifteen minutes. In 10 out of 17 pathological cases the head of the meal reached the cecum in one hour or less. Most "deficiency patterns" or more properly called "disordered motor function patterns" were associated with a delay in passage of the barium meal through the small intestine, and all cases of regional enteritis showed no appreciable delay.

REPORT OF CASES

Case I. Male, aged fifty. No clinical evidence of organic disease in gastrointestinal tract. This was a routine work-up. Small-intestinal study with the rapid method shows no evidence of disease and the head of the meal has reached the cecum in thirty minutes (Fig. 1, *A*, *B*, C).

Case II. Male, aged forty-three, with known duodenal ulcer for twenty years admitted because of abdominal pain, tarry stools, and clinical evidence of vitamin deficiency. Gastrointestinal series with rapid small bowel examination shows a deformed duodenal bulb with a niche. A "deficiency pattern" is demonstrated with the head of the meal in the cecum in sixty minutes (Fig. 2, *A*, *B*, *C*, *D*). The deficiency state was believed due to inadequate diet in the presence of long standing duodenal ulcer.

Case III. Male, aged fifty-seven, admitted because of weakness and copious, foul-smelling stools. Gastrointestinal series with rapid small bowel study shows abnormal small bowel pattern characterized by coarsening of mucosal folds, segmentation and dilatation with delay in transit time (Fig. 3, *A*, *B*, *C*, *D*). Laboratory studies established the diagnosis of non-tropical sprue (total fat in stool—37.6 per cent of dry weight). Patient was treated with vitamins, high caloric, low fat diet, liver extract, and folic acid with marked clinical improvement.

Case IV. Male, aged thirty-six, with anemia for two years for which he had a splenectomy at another hospital. No history of any gastrointestinal symptoms. Gastrointestinal series with rapid small intestinal study reveals constant filling defect in the proximal jejunum, interpreted as carcinoma (Fig. 4, *A*, *B*, *C*). Laparotomy was performed and an adenocarcinoma of the proximal jejunum was found.

Case V. Male, aged twenty-five, first admitted in 1946 because of diarrhea of three years' duration. Gastrointestinal series with small intestinal study by means of hourly films showed terminal enteritis. Under medical treatment he gained weight and diarrhea decreased. Gastrointestinal series using barium-water meal with small intestinal study by means of hourly films was done on June 2, 1947, and showed no appreciable change. Figure 5, *A*, *B* and *C* show the appearance of the small intestine at four, five and six hours. During the next six months, be showed no change clinically and received no additional treatment. Gastrointestinal series with rapid small intestinal study was done on January 5, 1948 (Fig. 5, *D*, *E*, *F*). The lesion is well demonstrated and spot roentgenogram (Fig. 5, *G*) shows the extensive involvement with polypoid changes in the mucosa of the terminal ileum.

DISCUSSION

By using a barium-normal saline mixture as the contrast medium followed by ice cold normal saline, we were able to obtain a marked decrease in the transit time of the meal through the small intestine without distorting the mucosal pattern. In many cases the mucosal pattern was more clearly delineated, and there was more complete filling of the entire small intestine than

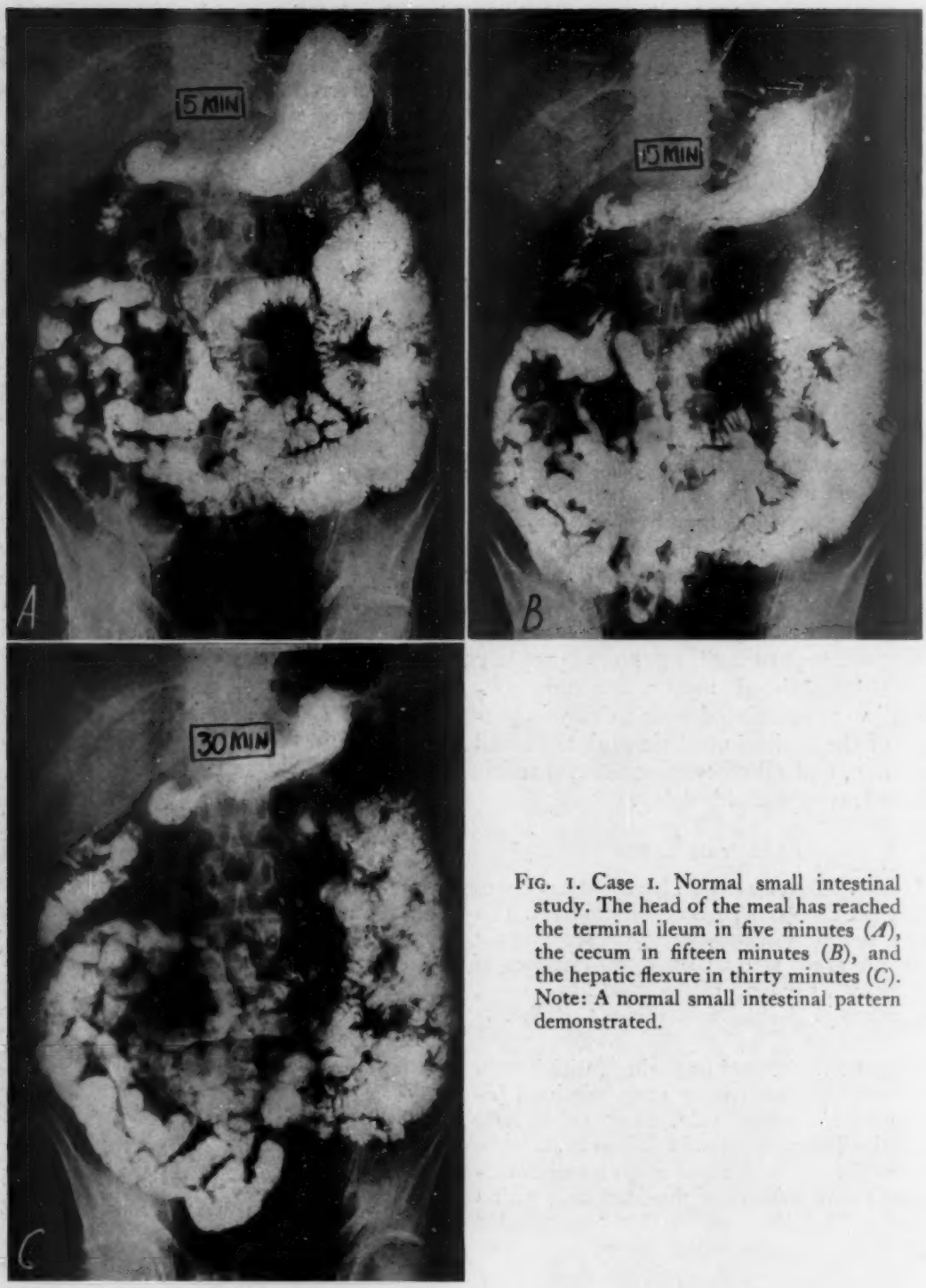


FIG. 1. Case 1. Normal small intestinal study. The head of the meal has reached the terminal ileum in five minutes (*A*), the cecum in fifteen minutes (*B*), and the hepatic flexure in thirty minutes (*C*). Note: A normal small intestinal pattern demonstrated.

that obtained by the hourly film technique. In 90 per cent of the normal cases studied, the examination was completed in one hour. In pathological cases, the rate of transit was also accelerated without sacrificing the appearance of the lesions.

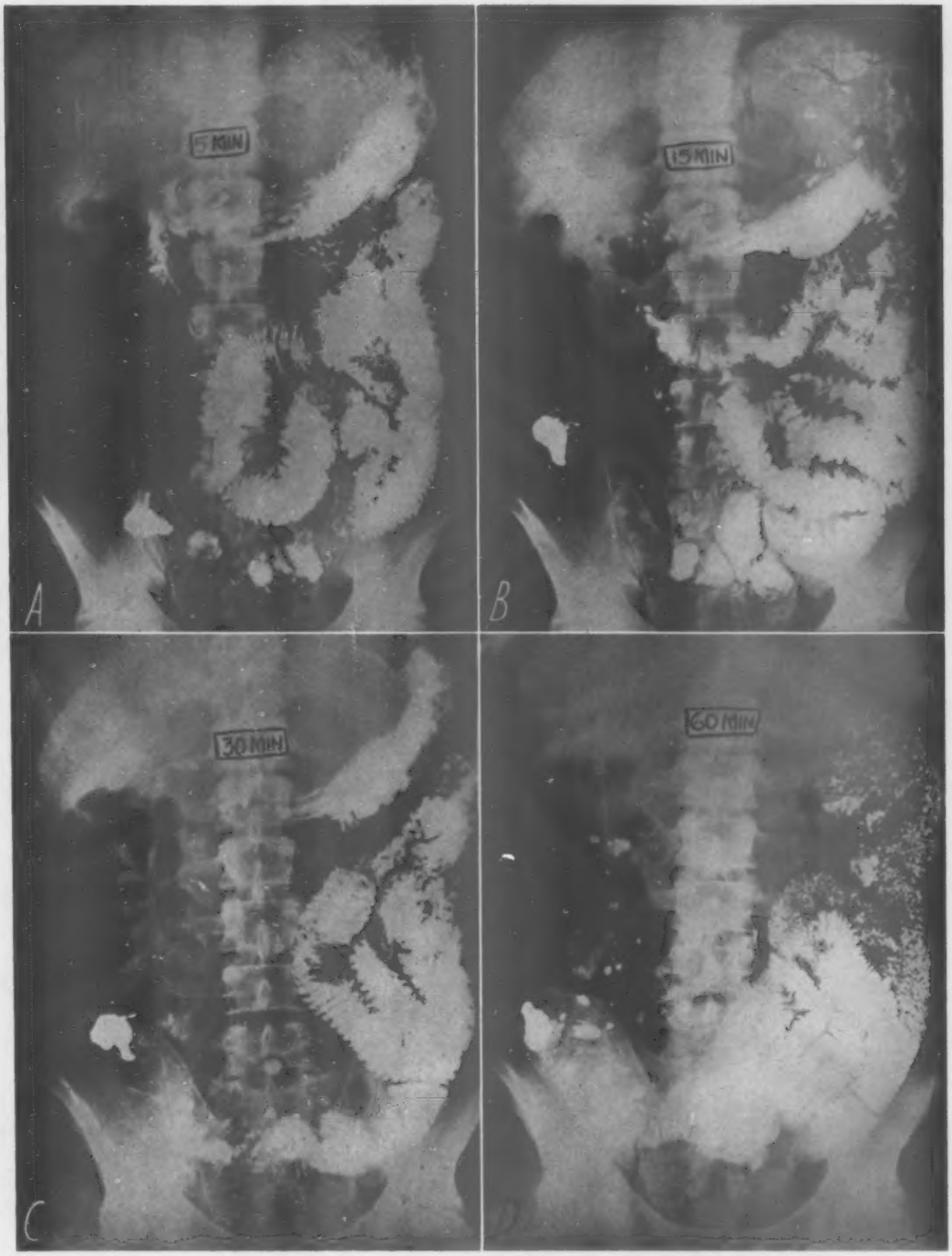


Fig. 2. Case 11. Chronic duodenal ulcer with activity and "deficiency state." The head of the meal is in the distal jejunum in five minutes (*A*), with little further progression of the meal in fifteen minutes (*B*) and thirty minutes (*C*). The head of the barium meal has reached the cecum in sixty minutes (*D*). Note: The delay in transit of the meal by standards of this method. The deficiency state is recognized by the abnormal pattern; coarsening of the mucosal folds, segmentation, dilatation and flocculation.

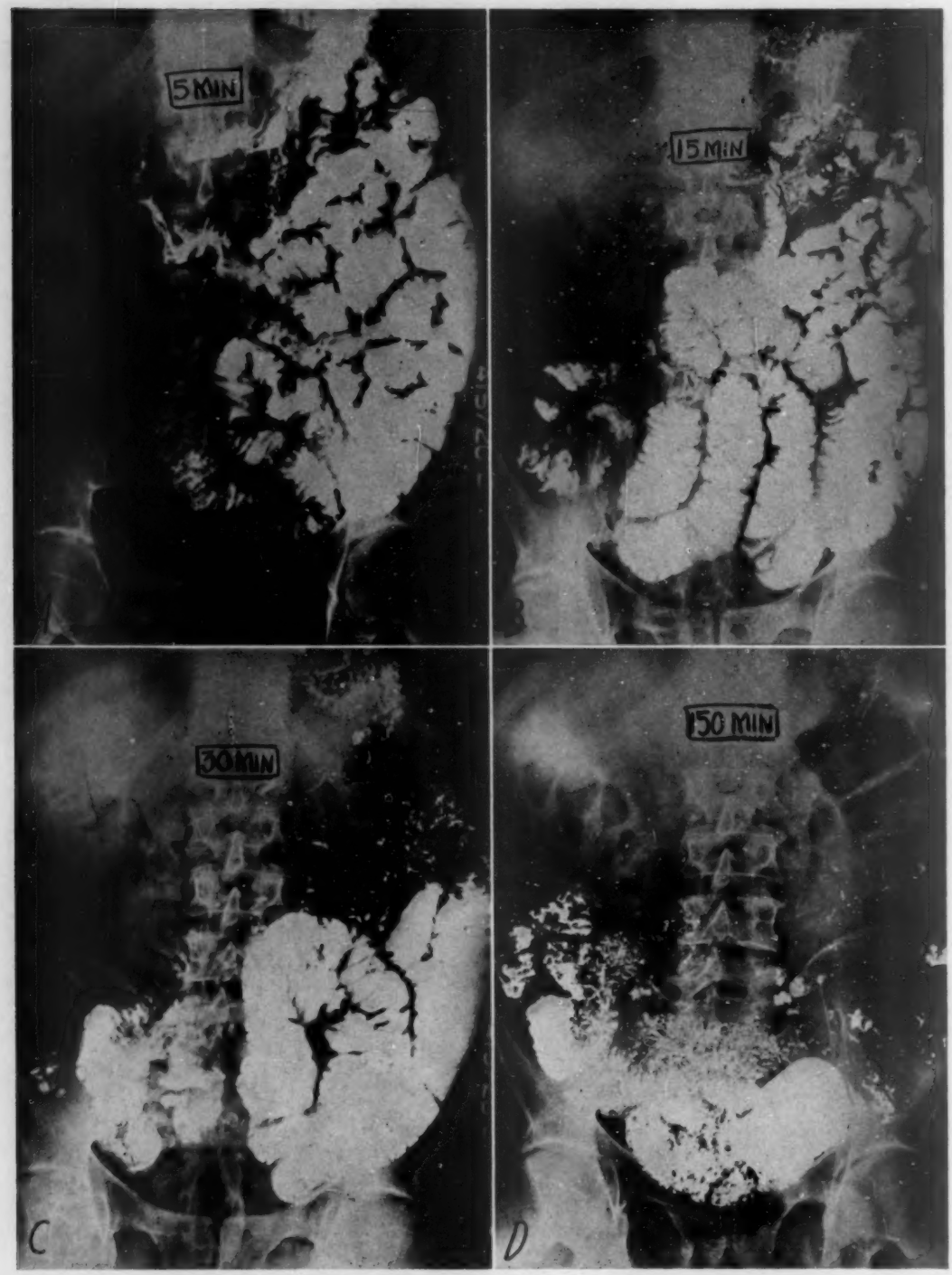


FIG. 3. Case III. Non-tropical sprue. The head of the meal is in the distal jejunum in five minutes (*A*), in the proximal ileum in fifteen minutes (*B*), and is still in the ileum at thirty minutes (*C*). The head of the meal has finally entered the cecum at 150 minutes (*D*). Note: The delay in transit of the meal by standards of this method, abnormal small intestinal pattern characterized by extensive dilatation, segmentation and flocculation most marked in the jejunum and proximal ileum.

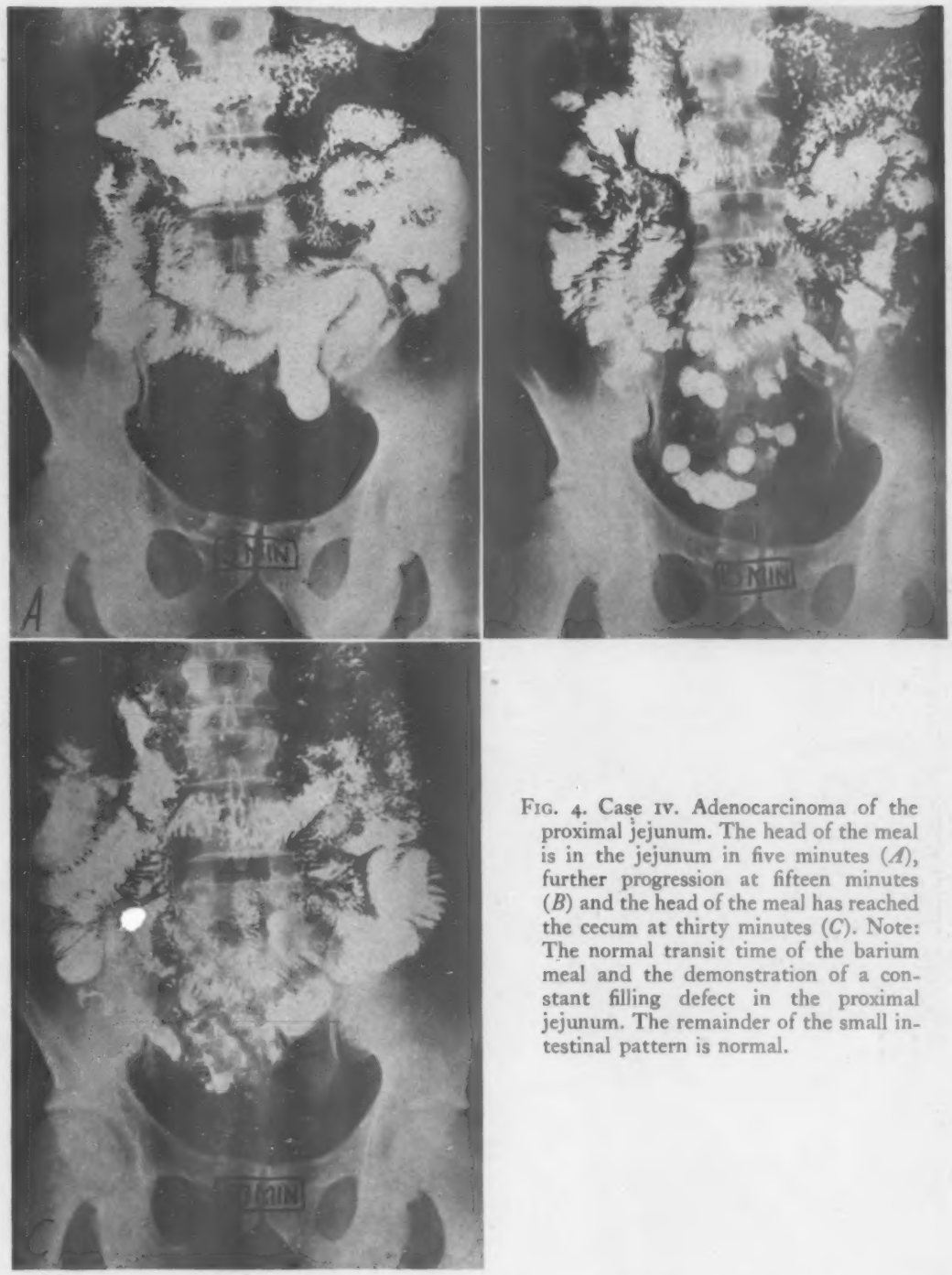


Fig. 4. Case IV. Adenocarcinoma of the proximal jejunum. The head of the meal is in the jejunum in five minutes (*A*), further progression at fifteen minutes (*B*) and the head of the meal has reached the cecum at thirty minutes (*C*). Note: The normal transit time of the barium meal and the demonstration of a constant filling defect in the proximal jejunum. The remainder of the small intestinal pattern is normal.

It is well known that in "deficiency states" or "disordered motor function patterns" and in sprue, there is a marked delay in the passage of the barium through the small intestine. We found this to be true with our method of examination, but the delay is only relative. For instance in one case of sprue (Case III), the head of the meal reached the cecum in 150 minutes. With the hourly method of examination it would

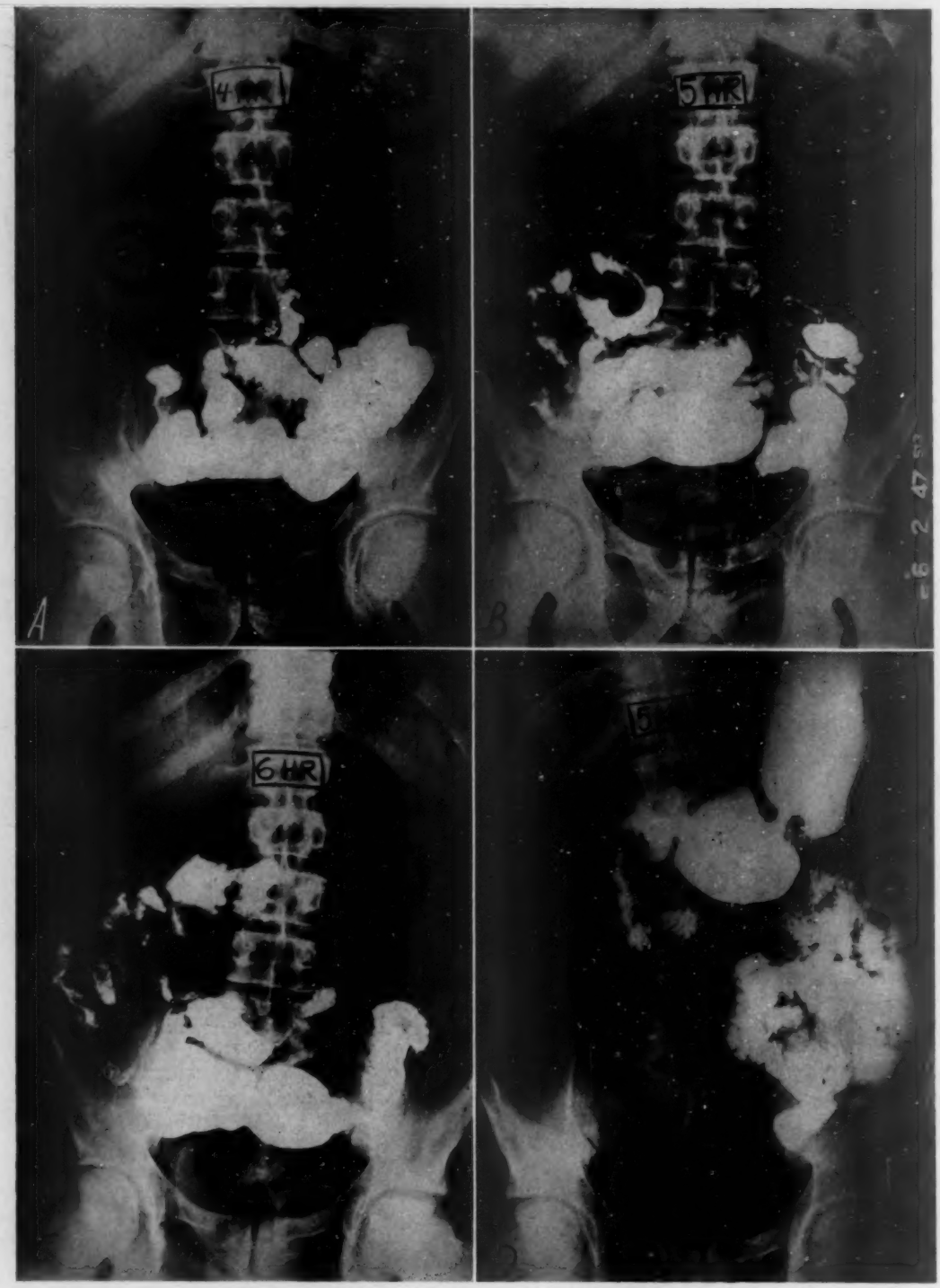
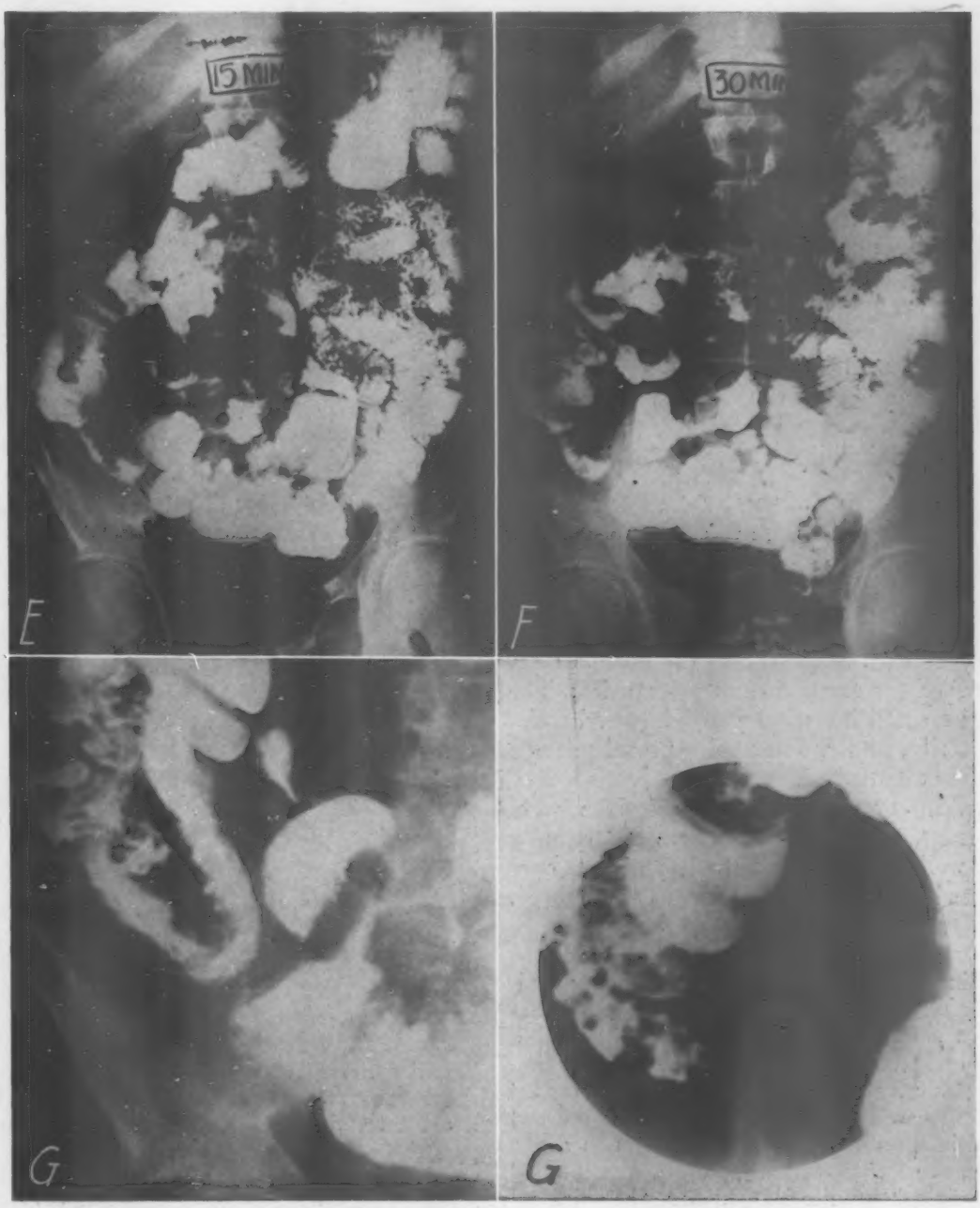


FIG. 5. Case V. Regional enteritis. (*A*), (*B*) and (*C*), four, five and six hour roentgenograms of examination of June 2, 1947. The head of the meal is in the transverse colon at six hours. (*D*), (*E*), (*F*) and (*G*), five, fifteen and thirty minute roentgenograms of examination of January 5, 1948, and spot roentgenograms of terminal ileum. The head of the meal has reached the descending colon at fifteen minutes and the rectum at



thirty minutes. Note: There is better demonstration of the pathological areas with the rapid method than with the hourly film method. The "string sign," and abnormal dilatation of the ileum are well shown on the fifteen and thirty minute films and on the spot films taken during fluoroscopy at thirty minutes. The fifteen minute films show further advance of the barium meal than does the six hour film.

have taken six to eight hours. Tumors of the small intestine can be well demonstrated with the rapid method (Case IV). In our cases of regional enteritis the lesions are better shown with the rapid method, and no delay in transit time is noted. In Case V, for example, the fifteen minute film of the rapid method is superior to the

six hour film of the hourly method.

The physiological basis of this rapid method has not been fully investigated as yet, but we feel that there are three main factors to be considered: (1) the accelerating effect of the increased mass of solution (total of 24 oz.) and the dilution of the barium meal; (2) the stimulation of peristalsis by a cold solution, and (3) the more physiologic action of a normal saline solution as compared to that of a water solution. With the more dilute barium mixture there is less dehydration and consequently less clumping of the barium and better filling of the small intestine, particularly of the terminal ileum.

SUMMARY

1. A rapid and practical method of roentgenologic examination of the small intestine has been presented.

2. In 90 per cent of the 87 normal cases studied with this method, the barium meal reached the cecum in one hour or less. The entire small intestine was delineated satisfactorily, and the normal mucosal pattern was not disturbed.

3. In 17 cases in which lesions were present, they were demonstrated equally as well with this method, and in some cases better than with the hourly technique.

ADDENDUM

Since submission of this paper further clinical studies with the rapid method showed that in 91 per cent of 235 normal cases the head of the meal reached the cecum in one hour or less. In 46 abnormal small intestinal cases the lesions were as well demonstrated with this method and in several instances were shown to better advantage than with the hourly film method Physiologic studies showed that the principal factor in this method is the action of a cold solution in relaxing gastric tonus and opening the pylorus. This accelerating action of a cold solution has been evaluated by Gershon-Cohen, Shay and Fels.[5]

New York Hospital
525 East 68th St.
New York 21, N. Y.

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DISCUSSION

Dr. Ross Golden, New York. Drs. Weintraub and Williams have presented a new method for the examination of the small intestine. They remark that there is no standard technique for the study of the small intestine. Many years ago, Pendergrass suggested 5 oz. of barium in distilled water as a standard barium meal. For him, this was a standard technique. We used it for a number of years, and then decided to try normal saline as a more logical medium from the physiological point of view, and through experience this has become standard for us. Dr. Pendergrass tried using normal saline and did not like it because he thought it made the transit time too short. What we like sometimes depends upon what we are used to. To me, it seems more important for each of us to use a technique the results of which we understand than for all of us to use exactly the same technique.

Drs. Weintraub and Williams compared their "rapid method" using cold saline to the distilled water medium at room temperature. It is not surprising that barium with cold saline goes through the intestine faster than barium with distilled water at room temperature because a saline preparation at room temperature goes through faster than barium in distilled water. Using this saline suspension at room temperature the barium reaches the cecum in one to one and a half hours in many cases. Inasmuch as Drs. Weintraub and Williams have their standards well set up for the cold saline method it would be interesting for them to compare it with the saline method at room

temperature. However, when barium enters the cecum, the examination is not necessarily over. The cecum is not always well filled at first and its appearance temporarily may not be normal. Furthermore, it is important to determine whether an apparent defect in any portion of the small intestine persists. Evidence of a disorder of the motor function of the intestine may be delayed. Furthermore, it is important to observe the effect of food on the intestine. After barium enters the cecum, we give a glass of milk. This is not only a food, but it is also one of the commonest of allergens. In a number of instances an overactive response has led to a suggestion that milk allergy might play a role in the clinical problem. If these points are borne in mind, I see no objection to the use of a large quantity of ice cold saline to hurry the barium along. We have used this Weintraub and Williams technique in a few cases and believe that it will probably prove in the long run to be a time saver. In cases of disordered motor function due to hypoproteinemia and edema, a tumor mass may not be outlined unless the barium is forced along rapidly and gives a continuous filling in spite of segmentation. This method may replace the small intestinal enema for this purpose.

THE PROCUREMENT AND CRITICAL APPRAISAL OF THE WIDTH DIAMETER OF THE MIDLINE RETROGASTRIC SOFT TISSUES

By M. H. POPPEL, M.D., F.A.C.R., A. SHEINMEL, M.D., *and* E. MEDNICK, M.D.
NEW YORK, NEW YORK

THE main purpose of this paper is to describe and to illustrate how the simple barium opacified stomach can be utilized to obtain the midline width of the retrogastric soft tissues if used in conjunction with the proper roentgen projections, posture and position.

In 1934 Engel and Lysholm described gastric pneumography. They filled the stomach with air or gas and took translateral films with the patient in the prone position. The air by rising would approximate the posterior wall of the stomach to the retrogastric soft tissues. Since that time the method has been used to a limited extent, either utilizing a small caliber stomach tube or effervescent powders. The stomach tube method, while unpleasant from the patient's standpoint, had the advantage of permitting withdrawal of residual stomach fluid. It also controlled the amount of air administered and permitted decompression if the patient became uncomfortable. Nevertheless, unintentional overdistention has been the cause of perforation of some existing ulcers. Often it was found that the air or gas failed to distinctly outline the posterior stomach wall and that the routine study with barium sulphate was interfered with.

Engel and Lysholm stated that the retrogastric soft tissues are equivalent in thickness to the anteroposterior diameter of the lumbar body at any level measured and that any increase in this thickness would be pathologic. Critical appraisal, however, even after applying proper correction for habitus, posture, height, weight, age, sex, respiratory state, stomach filling state and other possible variables, has convinced us that such is not true, because the posterior wall of the stomach varies considerably as regards its relationships. The cardia, and in some, a considerable portion of the body or even the entire pylorus, may be to the left of the midline lying in the left parasagittal region in the plane of the lower posterior ribs and musculature of the left lateral gutter. In such cases, in order to obtain the retrogastric soft tissue thickness, one would need a measurement to the posterior abdominal wall along the recessed gutter and not to the midline lumbar bodies.

We have abandoned the use of air and have substituted barium sulphate. This has necessitated that the patient's position be reversed from prone to the supine in order to bring the posterior wall of the stomach as close as possible to the spine, utilizing the weight of the barium in the stomach. Thus the passage of a stomach tube is obviated and the procurement of the routine films is not interfered with. Accordingly, our method consists of the administration of an ordinary barium sulphate mixture and the taking of translateral (left to right) and anteroposterior views. It is very important to fill the stomach completely and to obtain the exposures immediately, especially the translateral, in order to avoid gravitation of the barium into the cardia.

First, the translateral view is taken. Next without moving the patient and immediately after, in the same respiratory phase, the anteroposterior view is obtained. Thus a correlated biplane study is accomplished and errors due to change in position, posture, respiration and the state of stomach filling are avoided.

It is obvious that if any measurement is to be valid it must be taken from the posterior wall of only that portion of the stomach that crosses the midline to the anterior vertebral margin at the level of

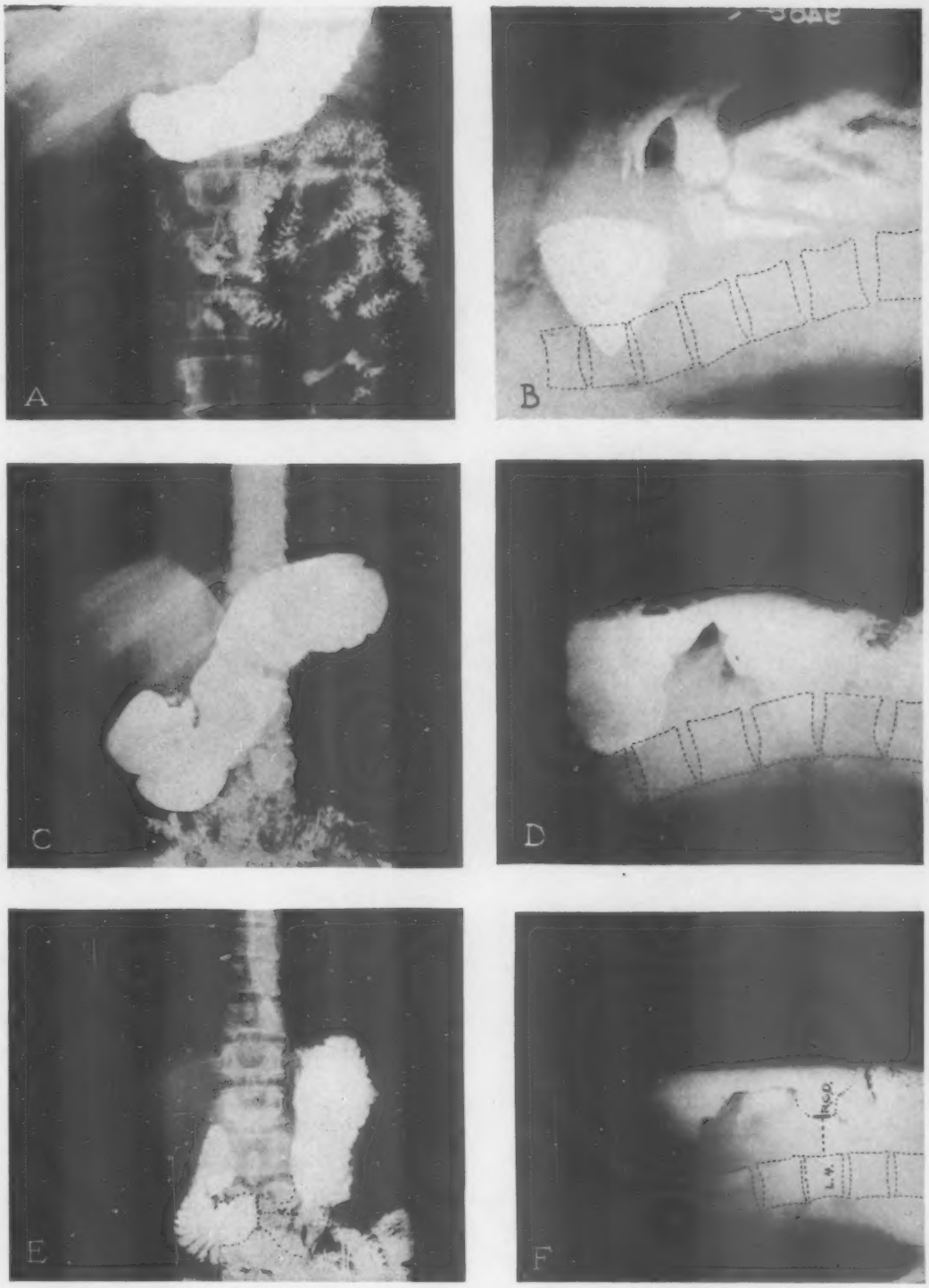

FIG. 1.* The various normal forms of habitus are illustrated in the anteroposterior and supine translateral projections: *A* and *B*, hypersthenic; *C* and *D*, sthenic; *E* and *F*, hyposthenic. These two films also illustrate how to obtain the vertical indicator (*V.I.*) by measuring the mid-distance between the lesser and greater curvatures of that portion of the stomach crossing the midline in the anteroposterior view. It is seen to be at the mid-level of the fourth lumbar body. This is then transposed to the exact vertebral level on the translateral and a perpendicular line from the anterior surface of the body to the nearest portion of the stomach is drawn. This is the retrogastric diameter (*R.G.D.*) in the midline.

* The illustrations were prepared by Mr. Sidney Shapiro.

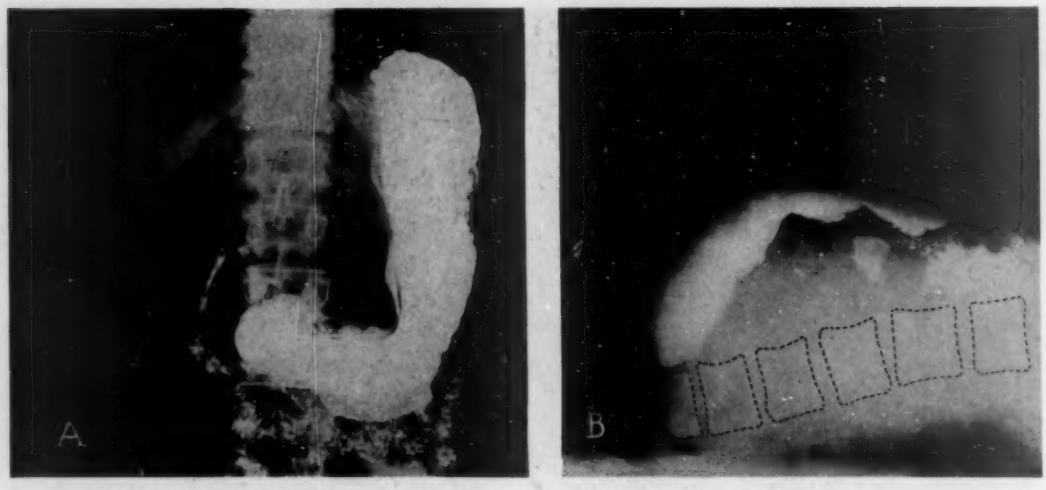

FIG. 2. This case illustrates the erroneous concept of measuring any other distance but the midline diameter. Ordinarily, this case would be considered abnormal, but measurement of the midline retrogastric diameter will show it to be normal.

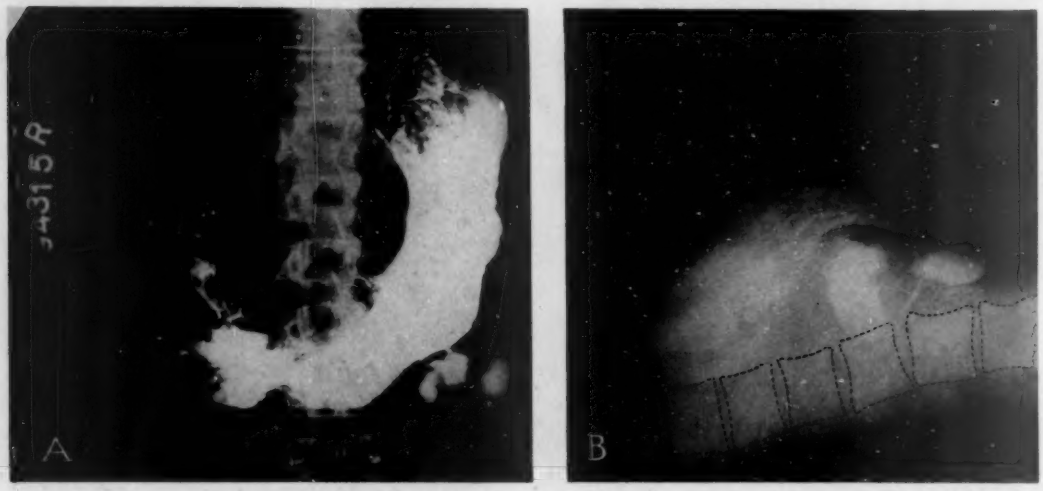

FIG. 3. This case shows an increase in the retrogastric midline soft tissue diameter due to cancer of the pancreatic head. Note the fistula from the prepyloric region to the pancreas.

crossing, otherwise the measurement would be an oblique one and it would be measuring the distance from a midline structure (vertebral body) to a portion of the stomach away from its anterior surface and therefore not related to it.

The anteroposterior view is the orientation film and is specifically taken to determine the exact vertebral body level at which any protion of the stomach crosses the midline. This is resolved to a point by mid-distance between the lesser and greater measuring the curvatures of that portion of the stomach crossing the midline. This point is called the vertical indicator. When it is applied to the translateral view one can accurately plot the midline retrogastric diameter at the proper vertebral level.

Now if the original postulate of Engel and Lysholm is applied it will be found to

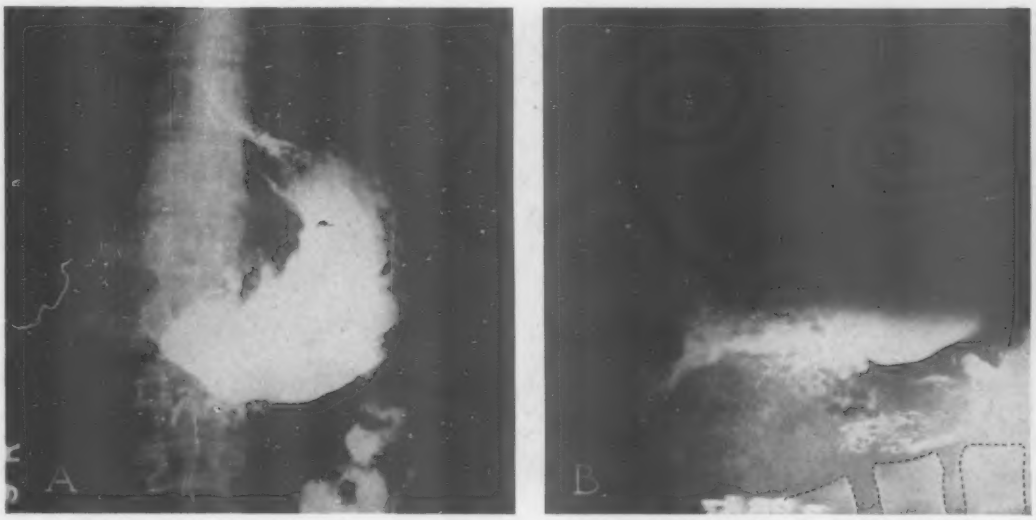

FIG. 4. This case shows no increase in the retrogastric midline diameter because the lesion does not possess any significant midline component. It does show an indenture upon the posterior wall of the stomach body and fundus, due to a cyst of the pancreatic body and tail.

be effective, i.e., "that the retrogastric soft tissues are equivalent in thickness to the anteroposterior diameter of the lumbar body at any level measured," provided one has ascertained what part of the stomach crosses the midline, at what vertebral level it crosses and then restricts the retrogastric measurement to this level only.

It must be borne in mind that the method is not infallible and that errors have been made. Holm found that extreme obesity, fat pads in the lesser sac and ascites may increase the retrogastric soft tissue measurement.

An increase in the midline retrogastric diameter cannot be translated into any one pathological entity in the absence of calcifications or other local distinguishing fea-

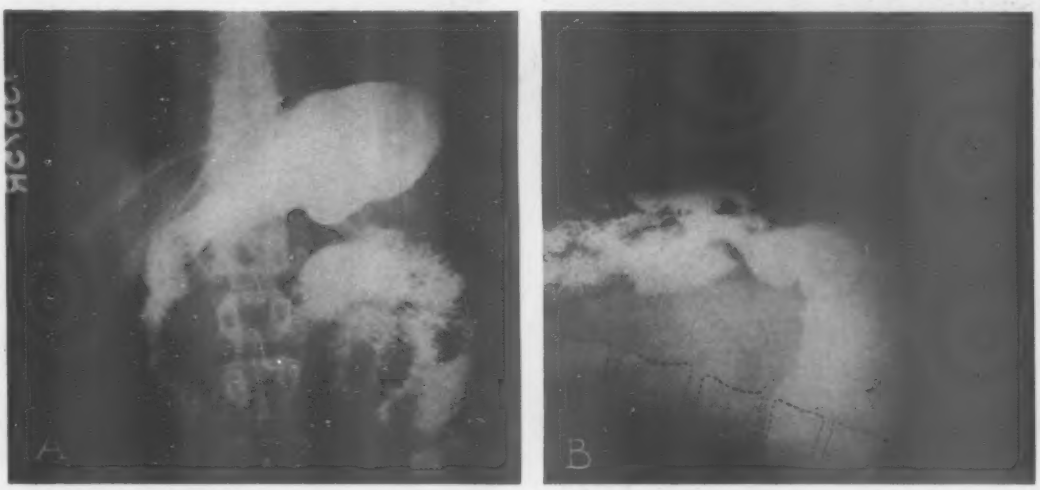

FIG. 5. This case shows an increase in the midline retrogastric diameter due to metastatic nodes from a seminoma of the testicle.

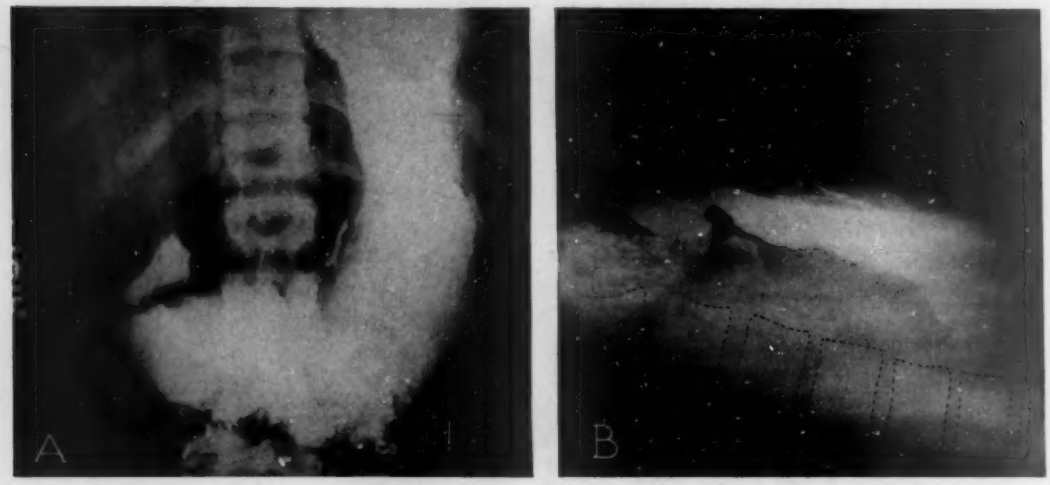

FIG. 6. This case of Hodgkin's disease shows multiple small indentures from enlarged retroperitoneal nodes upon the posterior wall of the body of the stomach, which is to the left of the midline. There is no increase in the retrogastric midline diameter because the lesion does not possess any significant midline component.

tures. The presence of disease elsewhere, as illustrated in Figures 5 and 6, aids in the correlation. Usually it simply indicates pathological enlargement of the normal regional structures (such as enlargement of the pancreas due to acute inflammation or tumor) or to the formation of adventitious extrapancreatic retrogastric mass or masses (such as retroperitoneal lymphosarcoma).

The corollary is also true that a normal midline retrogastric diameter does not exclude a retrogastric mass farther to the left. This is possible if the mass does not have any significant midline component.

At times it is not possible to obtain a midline retrogastric diameter for the simple reason that the stomach is entirely on the left. In such cases any attempt at measurement is obviously discouraged.

Likewise the presence of a localized, lobulated or generalized indenture on the posterior wall of any part of the stomach is significant in itself and does not have to be confirmed by measurements of any kind.

M. H. Poppel, M.D.
114 East 54th St.
New York 22, N. Y.

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BONE SCLEROSIS IN LEUKEMIA AND IN NON-LEUKEMIC MYELOSIS*

By FRANK WINDHOLZ, M.D., *and* SIDNEY E. FOSTER, M.D.
SAN FRANCISCO, CALIFORNIA

OSSEOUS manifestations of leukemia are mostly destructive lesions. In many instances, however, bone formation prevails instead of the absorption.

The significance of pathological bone formation in leukemia is but little understood. Although extensive investigations have been carried out on bone sclerosis associated with disturbances of blood formation, these studies have been more or less limited to cases of chronic non-leukemic myelosis. In this condition massive skeletal sclerosis is frequent. Records of bone sclerosis in monocytic, lymphatic and myeloid leukemias, and in aleukemic lymphadenoses are rare.[2,3,6,8,20,40,42]

Due to the scarcity of observations and because of the light which the knowledge of bone sclerosis in leukemia may throw on similar, still unexplained, bone changes in chronic non-leukemic myelosis, a further clarification of the subject seems warranted.

The diagnostic significance of the roentgen signs must also be studied. The presence of bone changes in living persons can be diagnosed only by roentgen examination. Roentgenological findings may be decisive in cases of leukemia where results of clinical and laboratory examinations are not characteristic. In aleukemic leukemia, bone changes are suggestive before the blood or clinical picture is diagnostic.

The purpose of this article is to present roentgenological findings of bone sclerosis in leukemia and aleukemic leukemia and in non-leukemic myelosis, and to correlate them with their clinical and patho-anatomical fundamentals in a further effort to clarify the subject.

MONOCYTIC LEUKEMIA

The study of bone changes in this disease is interesting not only because of their rarity (we found only 2 records of such occurrences), but also because of the biological difference of leukemic marrow in monocytic leukemia from that in all other forms of leukemia. Bone forming impulses of the medullary parenchyma in monocytic leukemia have to be investigated on the basis of this specific biological property of monocytic proliferations (see later). We observed 3 cases of monocytic leukemia with roentgenological densities in parts of the skeleton.

Roentgenograms of our first patient (Case I) showed increased density of both humeri and femora. (We shall refer to this distribution of bone changes as "polyostotic" sclerosis.) In addition, signs of periosteal bone formation were evident (Fig. 1*A*). In our second case (Case II) monocytic leukemia was associated with a cancer of the prostate and diffuse condensation of parts of the skeleton (Fig. 2). Microscopical examination revealed that many of the bone densities were consistent with bone forming tumor metastases. Several sclerotic areas, however, contained no metastases, but monocytic proliferations intermingled with fibrosis of the marrow. In our third case (Case III) roentgen signs of diffuse densities were seen in proximal portions of both humeri and in distal areas of both femora (Fig. 3).

Microscopic studies indicated that in monocytic leukemia pathological bone tissue develops in the medullary cavity as a direct metaplasia of intercellular reticulum or collagenous structures into woven membranous bone lamellae (Fig. 1*B* and 1*C*). The fibrils, preliminary to bone formation, are furnished by cellular activity of the leukemic tissue.[27] In this regard, bone formation in monocytic and, as we shall see later, in all other forms of leukemia, is dis-

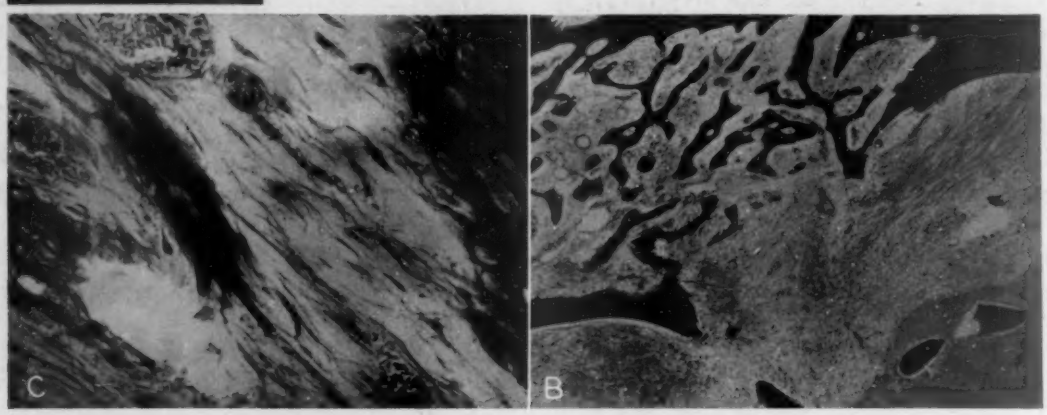
* From the Department of Medicine (Radiology), Stanford University School of Medicine, San Francisco, California.

tinctly different from bone formation in systemic and localized diseases, where bone tissue is laid down as the result of pathological activity of the osteogenic parenchyma (Paget's disease, hyperparathyroidism, bone cysts, osteogenic sarcoma, etc.). bone changes in the vicinity of destruction. Marchal, Mallet and Deprez[25] published the second recorded observation in 1945. Roentgenograms revealed large areas of bone destruction in the cranial vault. The authors considered their case a "forme os-

FIG. 1*A*. Case 1. Monocytic leukemia. Roentgenogram of right humerus showing thickening of cortex, periosteal bone shells, partial obliteration of the medullary cavity. B.W.F., male, aged eight weeks (No. 246224). Nodules on skin (monocytic infiltrates), hepatosplenomegaly and lymphadenopathy. Red blood cells 2.15 million, hemoglobin 6.42 gm. per cent; white blood cells 38,000, with 60 per cent monocytes, 2 per cent monoblasts, 12 per cent polymorphonuclears, 1 per cent eosinophils, 18 per cent lymphocytes, 1 per cent normoblasts per 100 white cells. Death following remarkable remission for six months.

FIG. 1*B*. Case 1. Photomicrograph ×70 from proximal metaphysis of right humerus. Van Gieson stain. Monocytic proliferation in right lower corner. Replacement of leukemic tissue by fibrous structures iu middle of photomicrograph. Destroyed old bone lamella on left lower side. Newly formed bone spicules in fibrous tissue in left upper and middle part of photomicrograph.

FIG. 1*C*. Case 1. Bone marrow in monocytic leukemia. Photomicrograph ×400, van Gieson stain. Leukemic tissue is replaced by reticular and collagenous fibers. In left upper corner and left center, thickened fibers are seen which gradually take on appearance of woven bone tissue. On right side apposition of new bone structures to older lamellae is seen.



Although bones in monocytic leukemia have not been subjected to detailed study, several authors[9,13,29] have written that bone changes may occur. To the best of our knowledge the first case was described by Kositchek[23] in 1943. Translucent bone lesions of a destructive character were seen in this case in many parts of the skeleton of a nineteen year old boy. Most translucent areas were surrounded by dense bony margins, indicating proliferating

seuse de leucémie chronique à monocytes." Biopsy taken from the area of destruction revealed, however, not monocytic but lymphocytic proliferations. The bone changes in this case need further clarification.

Our Case 11 is similar to the recently published findings of Bersack and Feinstein.[4] In their observation osteosclerosis in a case of cancer of the prostate was partly due to focal fibrosis in the marrow. The

patient was not afflicted with leukemia. Similar observation was made in Case VI of Sussman.[35] Here a malignant pleural mesothelioma was present with generalized metastases. Bone sclerosis was not due to metastases, but to fibrosis of the marrow.

Comparing our roentgenological findings in 3 cases of monocytic leukemia with those described in myelogenous and lymphatic forms, we believe the difference is obvious. Whereas in our cases of monocytic leukemia bone formation, even though minute, is in the foreground, in other leukemias bone proliferations are exceptional. This is understandable if one considers that monocytes as well as their derivatives and leukemic tissues contributed by them, exhibit the capacity to form intercellular reticulum, while lymphoid, myeloid and erythroblastic cells do not have this inherent property.[7,11,27] Since the presence of intercellular reticulum predisposes bone formation, it is understandable why bone sclerosis is more frequent in monocytic than in all other forms of leukemia.

The number of our observations is not large enough to permit generalizations as to what extent bone formation can be considered as a characteristic feature in monocytic leukemia. Yet our microscopical studies revealed that the tissue changes in the bone marrow, which are the precursors to bone formation, are regular constituents of the monocytic form of leukemic proliferations.

FIG. 3. Case III. Monocytic leukemia. Roentgenograms of lower ends of both femora revealed coarsening of spongy bone structures, thin cortex, increased density of medullary cavity and of lateral portions of both epiphyses. Dense transverse lines are seen in metaphyseal areas. W.T., aged three (No. A96992). Periods of lymphadenopathy, ecchymoses, hepatosplenomegaly, ulcers in mouth. Red blood cells 2.8 million, hemoglobin 44 per cent, white blood cells 27,000, with 18 per cent monocytes, 50 per cent monoblasts, 21 per cent immature stem cells, 3 per cent neutrophils and 7 per cent lymphocytes. Vital staining shows monocytic granules.

FIG. 2. Case II. Monocytic leukemia. Roentgenogram of ribs and spine. Diffuse sclerosis of eighth rib on left (see arrow). Microscopically fibrosis of marrow with monocytic proliferations. No tumor cells in this area, although patient has cancer of prostate with bone metastases. H.N., aged eighty-five (No. 27740). Prior, treated for cancer of prostate. Skin infiltrates, ulcers in mouth. Blood count: red blood cells 2.3 million, hemoglobin 34 per cent; white blood cells 66,000 with 40 per cent polymorphonuclears, 12 per cent lymphocytes, 37 per cent monocytes, and 10 per cent monoblasts. At autopsy extensive monocytic infiltrates in liver, spleen, lymph nodes, hypophysis, kidney, skin and bone marrow. Many of bony densities are tumor metastases, others (eighth rib) caused by bone formation in fibrous monocytic marrow.

It is also remarkable to realize the difference in bone changes in leukemic and aleukemic forms of monocytic leukemia. Whereas in lymphatic and myelogenous leukemias the aleukemic forms exhibit a greater tendency toward production of bone (see later), it seems that osseous changes in aleukemic reticulosis (aleukemic form of monocytic leukemia) are more frequently destructive. Further observation reveals, however, that in certain conditions bone forming properties of monocytic and histiocytic proliferations can also be demonstrated in the presence of bone absorption in hyperplastic and neoplastic lesions of aleukemic reticulo-endotheliosis.[39,40] Feher[14] presented skull roentgenograms of a child suffering from aleukemic reticulosis. A small, highly radiosensitive area of bone destruction was exhibited. However, most of the bones of the vault revealed diffuse thickening of spongy bone lamellae, producing roentgen signs of sclerosis of the diploë.

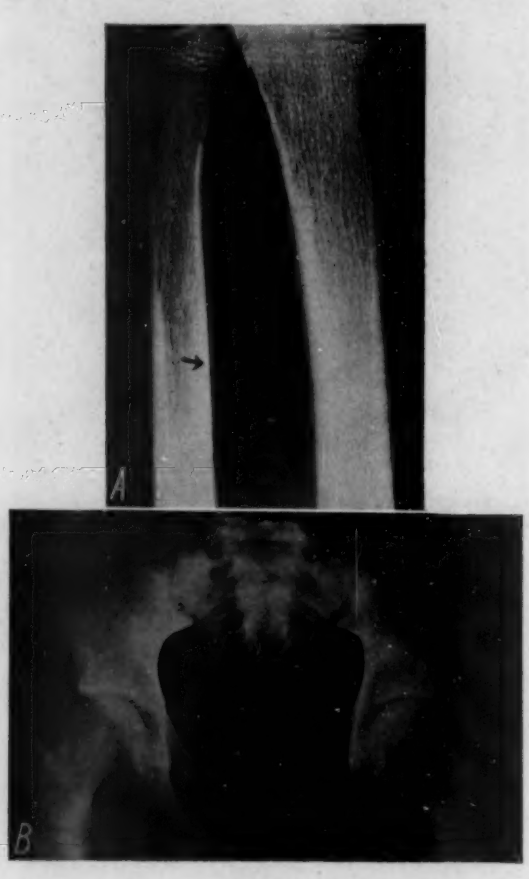


FIG. 4*A*. Case IV. Lymphatic leukemia. Roentgenograms of lower left forearm (slightly enlarged) show coarsening and thickening of spongy structures and cortex. Faint periosteal ossification on radial aspects of the cortex of radius (see arrow).

FIG. 4*B*. Case IV. Bones of the pelvis are denser than in normal conditions. Structure is somewhat irregular due to narrowing of marrow spaces and thickening of bony trabeculae. W.G.P., male, aged sixteen (No. 216040). Muscle and joint pains, splenomegaly, lymphadenopathy. Red blood cells 3.7 million, white blood cells 45,000, with 1 per cent neutrophils, 1 per cent eosinophils, 3 per cent myelocytes, 17 per cent lymphocytes, 78 per cent lymphoblasts. No autopsy.

LYMPHATIC LEUKEMIA

Roentgenological signs of bone changes exhibited in lymphatic leukemias are caused as a rule by destructive lesions. They consist of pin-point, punched-out areas in the cortex of long bones and skull, erosions at the inner aspect of the cortical layers, and transverse rarefied areas near the epiphysis. Occasionally, periosteal ossification is associated with the above changes.

Signs of bone formation are more frequent in aleukemic forms of lymphatic leukemia than in leukemic forms.[6,31,41] Bone sclerosis is very rare in the presence of leukemic lymphatic leukemia.[2,3,6,18,42]

We had the opportunity to study 5 cases of lymphatic leukemia with proliferative bone changes. In one of these instances a true leukemia was present. The four others were aleukemic lymphadenoses.

In the case of the leukemic lymphatic leukemia (Case IV), the entire skeleton was denser than normal (Fig. 4*A* and 4*B*). We propose to denominate such distributions of bone sclerosis as "panostotic" sclerosis. Irregularities of cancellous structure, spotty condensations, and coarsening of dense trabeculae were seen in vertebral bodies, pelvis (Fig. 4*B*), ribs, and long bones. Periosteal bone formation was displayed at the distal end of the left ulna (Fig. 4*A*) and of the right humerus.

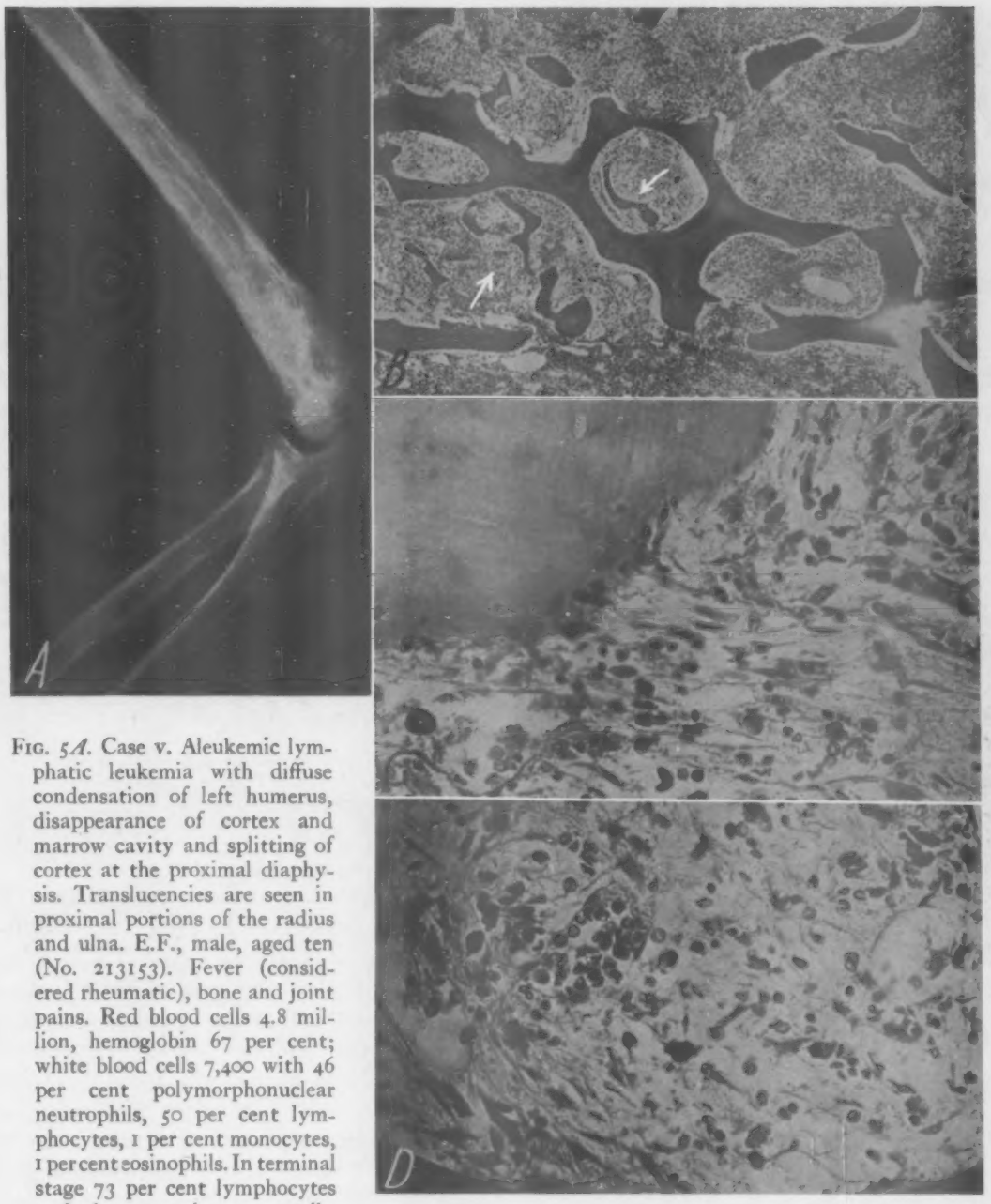


Fig. 5*A*. Case v. Aleukemic lymphatic leukemia with diffuse condensation of left humerus, disappearance of cortex and marrow cavity and splitting of cortex at the proximal diaphysis. Translucencies are seen in proximal portions of the radius and ulna. E.F., male, aged ten (No. 213153). Fever (considered rheumatic), bone and joint pains. Red blood cells 4.8 million, hemoglobin 67 per cent; white blood cells 7,400 with 46 per cent polymorphonuclear neutrophils, 50 per cent lymphocytes, 1 per cent monocytes, 1 per cent eosinophils. In terminal stage 73 per cent lymphocytes and 26 per cent immature cells, thought to be lymphoblasts. Autopsy: Many organs, including liver and spleen, are infiltrated by cells in appearance of larger lymphocytes. Many ulcers were found in the intestinal tract and a cardiac malformation was present.

Fig. 5*B*. Case iv. Fibrous aplasia of bone marrow. Photomicrograph ×75. Hematoxylin-eosin. The marrow cavity is filled by fibrous tissue contributed by fibrocytes, intercellular collagenous fibers, capillary blood vessels, mast cells, small groups of lymphocytes. Metaplastic bone formation is indicated by arrows. A few fat cells are seen in left lower corner.

Figs. 5*C* and 5*D*. Case iv. Photomicrographs ×400. Hematoxylin-eosin. Fibrous marrow in aleukmic lymphatic leukemia. Numerous spindle shaped fibrocytes and intercellular fibers seen in close relationship to newly formed (pink) bone structures in left center. Isolated lymphoid cells, megakaryocytes, and small cell groups are spread over the section.

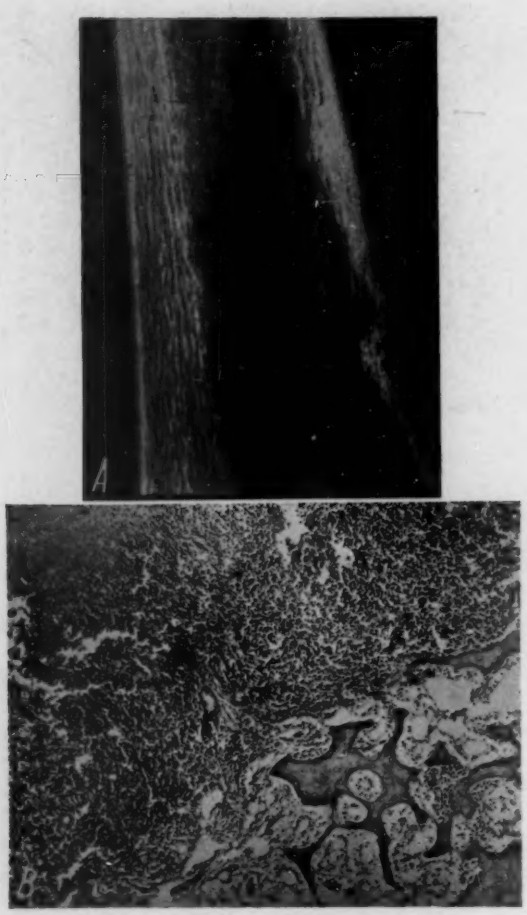


FIG. 6*A*. Case VI. Aleukemic lymphatic leukemia. Roentgenogram of the distal end of the left femur (slightly enlarged), showing thickening of trabecular structure and bony proliferation in inner aspect of the cortex (on right). T.C., male, aged sixteen (No. 230239). Weakness, fever, dyspnea. Red blood cells 1.1–3.47 million, hemoglobin 20–60 per cent; white blood cells 600–2,900 with 81–88 per cent lymphocytes and many primitive lymphoblasts. Autopsy: Spleen, liver, retroperitoneal nodes, bone marrow extensively infiltrated with lymphocytes; many necroses are seen in proliferating lymphatic leukemic tissue.

FIG. 6*B*. Case VI. Aleukemic lymphatic leukemia. Photomicrograph ×75 from the lower end of the femur (hematoxylin-eosin). Marrow parenchyma replaced by leukemic infiltrates. Necrosis in leukemic proliferations in left upper corner and considerable increase of intercellular collagenous structures in center of photomicrograph. Metaplastic bone formation partly within increased intercellular fibers in right lower corner of photomicrograph.

The roentgen appearances of sclerosis in our 4 observations of aleukemic lymphatic leukemia were various.

(a) Sclerosis of a single bone (monostotic sclerosis) with splitting of cortex. Bone destruction in other bones (Fig. 5*A*) (Case V).
(b) Bony proliferations on the inside of the cortex of the femur, thickening of spongy structures at the femoral metaphysis (Fig. 6*A*) (Case VI).
(c) Sclerosis of the diploic structures on the skull intermingled with small translucencies (Fig. 7*A*) (Case VII).
(d) Circumscribed areas of bone formation in the medullary cavity of the ulna and periosteal ossification over the cortex of the femur (Fig. 8) (Case VIII).

Microscopical examination was carried out in 2 cases (V and VI) of aleukemic lymphadenosis, and in 1 case (VII) an enlarged lymph node was studied microscopically.

The histopathological appearance of osteogenesis was similar to that in monocytic leukemia. Here, too, bone spicules were formed as direct metaplasias of collagenous and reticular interstices into woven bone structures. As a result of this bone formation, the amount of spongy structures was increased.

In the sclerotic right humerus (Case V), the entire marrow parenchyma was replaced by a vascular loose fibrous tissue containing only small groups of isolated leukemic cells. The fibrous tissue was most dense about capillary blood vessels and near the cortex at the endosteum. It seemed to originate in adventitial cells of perivascular areas. There were microscopical indications that the collagenous structures were not produced by leukemic cells themselves as in monocytic leukemia (Fig. 5*B*, 5*C*, 5*D*).

In our Case VI the leukemic proliferations in the bone marrow revealed multiple areas of necrosis. In the surroundings of such necrosis, the intercellular collagenous fibers were increased and seemed to give rise to the development of bony proliferations (Fig. 6*B*).

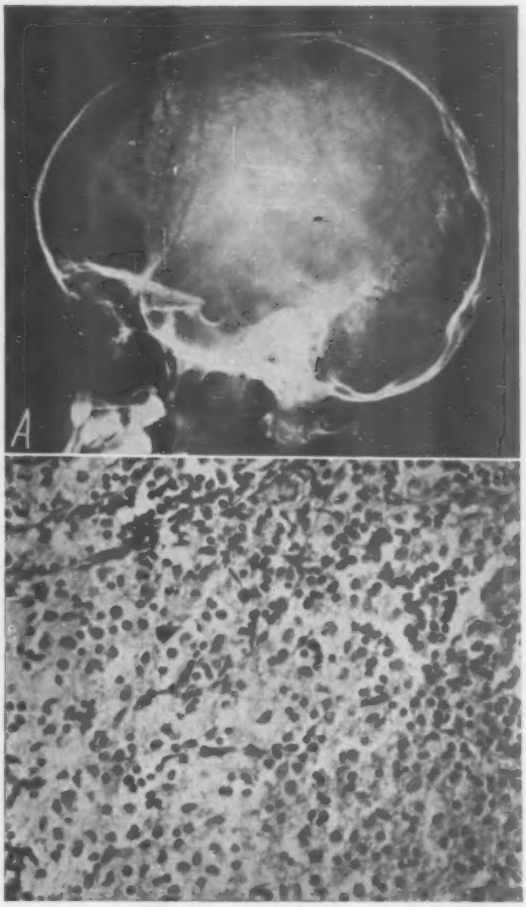


FIG. 7*A*. Case VII. Aleukemic lymphatic leukemia. Lateral roentgenogram of skull showing increased density of diploic structures alternating with faint translucencies in parietal areas of cranial vault. B.M., female, aged eight (No. 222985). Headaches, pallor, fatigue, generalized lymphadenopathy, slight hepatosplenomegaly. Red blood cells 1.5–2.9 million, hemoglobin 30–55 per cent, white blood cells 2,920–4,000 with 75–77 per cent lymphocytes, many of them immature, 22 per cent neutrophils, 3 per cent monocytes, 26,250 platelets. Lymph node biopsy shows reticular hyperplasia. No autopsy.

FIG. 7*B*. Case VII. Aleukemic lymphatic leukemia. Photomicrograph ×250 of enlarged axillary lymph node, showing proliferation of large translucent cells of reticular origin (left lower part of section) in appearance of diffuse reticular hyperplasia. Few lymphoid cells are scattered throughout the field.

FIG. 8. Case VIII. Aleukemic lymphatic leukemia. Roentgenogram of the left elbow showing thickened cortex of the ulna and circumscribed areas of bony proliferation, obliterating a part of the medullary cavity. R.K., male, aged sixty-seven (No. 252020). Aplastic type of anemia for five years, joint pains, hematuria, lymphadenopathy, splenomegaly. Sternal biopsy shows numerous lymphoblasts. Red blood cells 2.8 million, hemoglobin 40 per cent; white blood cells 3,800 with 46 per cent neutrophils, 2 per cent eosinophils, 50 per cent lymphocytes, 2 per cent lymphoblasts. No autopsy.

Only an enlarged lymph node could be studied in Case VII. It revealed a proliferation of reticular cells intermingled with infiltrations of lymphocytes (Fig. 7*B*). Whether this appearance of the lymph node permits us to draw a conclusion as to the probable appearance of the bone marrow cannot be stated. In uncomplicated lymphatic leukemia both lymph nodes and bone marrow are simultaneously infiltrated by lymphocytes. However, in early stages of leukemic proliferations reticular hyperplasia may prevail (Stasney and Downey).

When one reviews the clinical data of our observations of lymphatic leukemia, it is obvious that no relationship exists between the age of the patient, the blood count, the duration of the disease, and the type of bone changes present. The microscopical appearances were very obvious and persistent. In places in which roentgenograms revealed proliferative bone changes, microscopical study disclosed the presence of focal fibrosis or of fibrous transformation of the bone marrow of an entire bone. This is an important finding because the general opinion[31] has been that in cases of lymphatic leukemia no direct increase in connective

tissue is observed, except in the bone marrow following radiation therapy. In bones which did not exhibit osseous proliferation, the fibrosis was absent and the medullary cavity contained hemopoietic parenchyma, leukemic infiltrations, or fat or a combination of these structures.

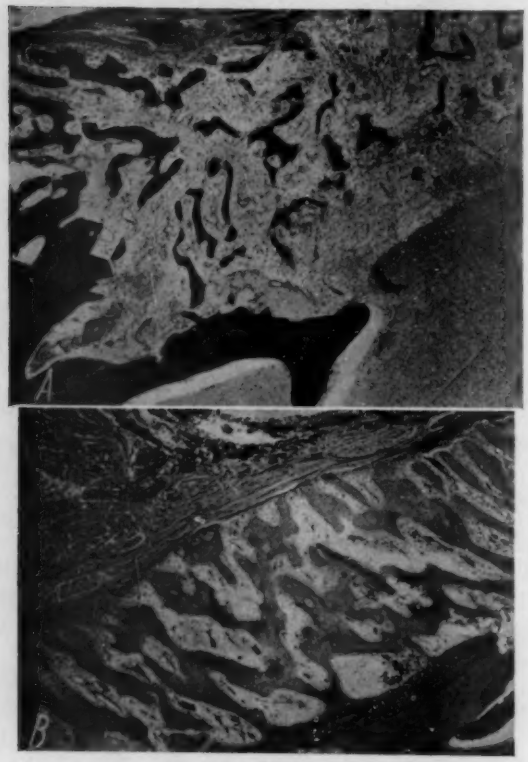


FIG. 9*A*. Case I. Periosteal bone formation in monocytic leukemia. Photomicrograph ×75. Van Gieson stain. Cortex of humerus. Leukemic proliferation in right lower corner invades and destroys bone lamellae of the cortex. Destroyed area is filled by fibrous tissue, residues of leukemic proliferations and newly formed bone lamellae in appearance of callus. Periosteum in left upper corner of section. There is no "lifting" of the periosteum seen by leukemic cells.

FIG. 9*B*. Case V. Pericsteal bone formation in aleukemic lymphatic leukemia. Photomicrograph ×75. Hematoxylin-eosin stain. Old cortex with leukemic cells in Haversian canals, right lower corner. Perpendicular bone lamellae having the appearance of callus, in middle of picture. Periosteum in left upper corner.

THE SIGNIFICANCE OF PERIOSTEAL BONE FORMATION IN LEUKEMIA

We observed this form of bone proliferation in 4 cases. In Case I, monocytic leukemia, periosteal osteogenesis was seen in most of the bones exhibiting roentgenographic densities. In Case V, lymphatic leukemia, in which the entire skeleton was denser than normal, periosteal ossification was limited to the distal end of the left radius and of the right humerus. In Case VI, aleukemic lymphatic leukemia, periosteal bone formation was intermingled with newly formed bone structures in the medullary cavity. Case VII revealed periosteal bone shells at the distal end of the left femur. In this area minute translucencies of the cortex (destructions) were seen just below the periosteal bone reaction. No translucent areas were seen in other parts of the femur.

Microscopical studies were done in Cases I and VI. They revealed that the roentgenologically seen periosteal bone shells are furnished by perpendicularly and obliquely oriented bone spicules on the outer surface of the cortex (Fig. 9*B*). (See also Kalayjian, Herbut and Erf.[21]) They developed, in our cases, in areas in which the cortex had been previously destroyed by leukemic tissues (Fig. 9*A*). Osteogenesis followed the events which take place in callus formation after injuries to bones. The callus in turn is replaced by fibrous structures furnishing the matrix of osseous metaplasia.

We failed to observe a true lifting of the periosteum by a subperiosteal accumulation of leukemic cells. This was assumed by earlier investigators.[30,35] It seemed to us that the periosteum did not present more of an obstacle to the infiltration of leukemic cells than did bone or muscle tissue. They invaded the periosteum with the same apparent ease with which they invaded the Haversian canals, perivascular spaces, bone tissue, cartilage and muscle tissue outside the periosteum. Leukemic cell groups found between newly formed periosteal bone

tissue and the cortex were in all cases small and did not impress us as being massive enough to lift the periosteum mechanically. We had to consider periosteal bone shells, in our cases, as being caused by mechanical influences (see also Caffey[6]) which followed weakening of the cortex by a large number of minimal destructions. In Case VII, such minute destructions were seen on roentgenograms as being limited to the areas of periosteal ossifications.

MYELOID LEUKEMIA

The estimation of the frequency of association of myeloid leukemia with sclerotic bone changes is difficult because in references, in many instances, non-leukemic chronic myelosis is described as myeloid leukemia.[35] In this condition bone sclerosis is frequent (in one-third of the cases). It seems, however, that there are only a few cases on record in which leukemic myelosis was associated with more or less extensive sclerosis of bones.[8,18,35] All the other observations published as bone sclerosis in myeloid leukemia were closer to non-leukemic chronic myelosis than to myeloid leukemia. In our series of 52 cases of chronic myeloid leukemia, we found destructive bone lesions 8 times. We observed no sclerosis.

The relative scarcity of observation of proliferative bone changes in myeloid leukemia is difficult to explain, if one considers that non-leukemic forms of chronic myelosis reveal rather frequent and, at times, most extensive panostotic forms of sclerosis. It is, however, reasonable to believe that in true myeloid leukemia the precursor of osteogenesis, namely, fibrosis, develops rarely. The bone marrow in this disorder usually reveals a remarkable hyperplasia of all myeloid cells. There is a more pronounced shift towards immaturity displayed than toward formation of histogenous cellular derivatives,[15] specifically collagenous and reticular fibers.

In brief, it seems to us that the reason why productive bone changes are seen so rarely in true myeloid leukemia lies in the failure of its cells to contribute to the formation of fibrous structures, and in genetic differences which are characterized by a greater tendency toward immaturity than is observed in lymphatic or monocytic leukemia. In rare instances, however, osteogenesis may occur.

Fibrosis in the marrow without sclerosis in leukemic myelosis has been repeatedly described.

CHRONIC NON-LEUKEMIC MYELOSIS

This disease differs in many features from the leukemic form of myelosis. It is characterized by a low leukocyte count, long duration, extraordinary enlargement of the spleen, immature cells of myeloid series in the peripheral blood, and an absence of the characteristic blood picture of myeloid leukemia. This condition has been associated with massive sclerosis of the skeleton in more than one-third of the observations. Such cases have been described under a large variety of denominations,* depending upon association of the disease with only fibrosis of the bone marrow or with fibrosis of the marrow and sclerosis of the bone.[1,2,19] Variation of the blood picture has also given additional incentive for variations of classification.[41] In general, cases are rare. Up to 1943, only 75 cases were listed.[32] Since that time, a number of new observations have been reported.[35] Hematological and pathological problems associated with these questions are of great complexity.

We had the opportunity to observe 2 cases of chronic non-leukemic myelosis associated with osteosclerosis in panostotic distribution.

The first observation (Case IX) is that of a male, aged sixty-two, (No. A98842). Splenomegaly was established thirty years ago. The first blood count revealing the patient's condi-

* Leuko-erythroblastic anemia with myelosclerosis (Mendeloff and Rosenthal), osteosclerosis with myelofibrosis, myelosclerosis (Vaughan), aleukemic myelosis with osteosclerosis (Hirschfeld), osteosclerotic anemia, myelosis with osteosclerosis (Hewer), myelophthisic splenomegaly (Ballin and Morse), myelosclerosis associated with leukemoid blood picture (Mettier and Rusk), chronic non-leukemic myelosis (Hickling), leukanemia (Leube and Arneth), etc.

FIG. 10. Case IX. Chronic non-leukemic myelosis. Roentgenogram of the left femur (taken 1945) showing diffuse bone sclerosis in neck and isolated areas of sclerotic foci in intertrochanteric areas. Male, aged sixty-two (A98824). Splenomegaly and anemia for thirty years (further data in text).

tion dates back fifteen years. Eight years ago an episode of polycythemia vera developed which was checked after eight months' duration by moderate doses of roentgen rays to the spleen. Characteristics which distinguished his blood picture from that of myeloid leukemia were: erythroblastosis out of proportion to the degree of anemia, and a total leukocyte count which was not as high as that seen in leukemia. At the same time the morphological pictures showed values seen in leukemia. Platelets were reduced in number and not increased as seen in myeloid leukemia with this type of morphological picture. The blood count remained essentially the same with the exception of the episode of polycythemia,* with a progressive enlargement of the spleen until the most recent examination in 1946. Comparative blood counts are recorded in Table I.

The first roentgen examination in this case was made in 1940, twenty-three years following the discovery of an enlarged spleen. At that time the roentgen appearance of the examined bones was normal (Fig. 11*C*). This, however, does not indicate that pathological changes were absent. Vaughan describes a case of panostotic sclerosis in which histopathological changes of osteosclerosis were seen, despite negative roentgen findings.

Four years later in our case, irregular densities developed in the spine, upper humeri, femora and pelvis (Fig. 10, 11*A*). They revealed remarkable progress during the following year, exhibiting finally characteristic features of massive panostotic sclerosis (Fig. 11*B*, 11*C*).

Microscopical examination of a rib (Case IX) which had been biopsied in 1944 (ST 56–756) showed a greatly thickened cortex with increase in the number and dimensions of the spongy bone trabeculae. Most of the trabeculae revealed homogeneous osteoid pink borderlines. They were frequently fused with connective fibers which filled the narrow medullary spaces. The reticular stroma appeared to originate in

* The possibility of association of erythremia with leukemia and aleukemic myelosis has led to the publication of a number of case reports[17] purporting to demonstrate this relationship. In most cases, the amount of leukemic tissue greatly exceeded the amount of erythroblastic structures, and all the pathological findings greatly resembled those characteristic of chronic myeloid leukemia.[34,35] The sequence of events, namely the polycythemia developing in a case of typical leukemia, has also been noted. Ghiron[18] observed a case in which during the course of typical myeloid leukemia the erythrocyte count increased from 2.4 million to 7.2 million and, as in our case, ruddy discoloration of the face developed.

TABLE I

Year	Red Blood Cells	White Blood Cells	Polymorpho-nuclears	Myelo-cytes	Myelo-blasts	Lympho-cytes	Plate-lets	Reticulo-cytes	Normoblasts
1930	5.0 million	19,875	70%	25%	—	17%	395,000	3.0%	5 per 400 L.
1940	8.4 million	23,000	63%	6%	8%	8%	193,000	2.4%	16 per 400 L.
1946	4.9 million	15,000	64%	17%	—	12%	12,000	—	12 per 400 L.

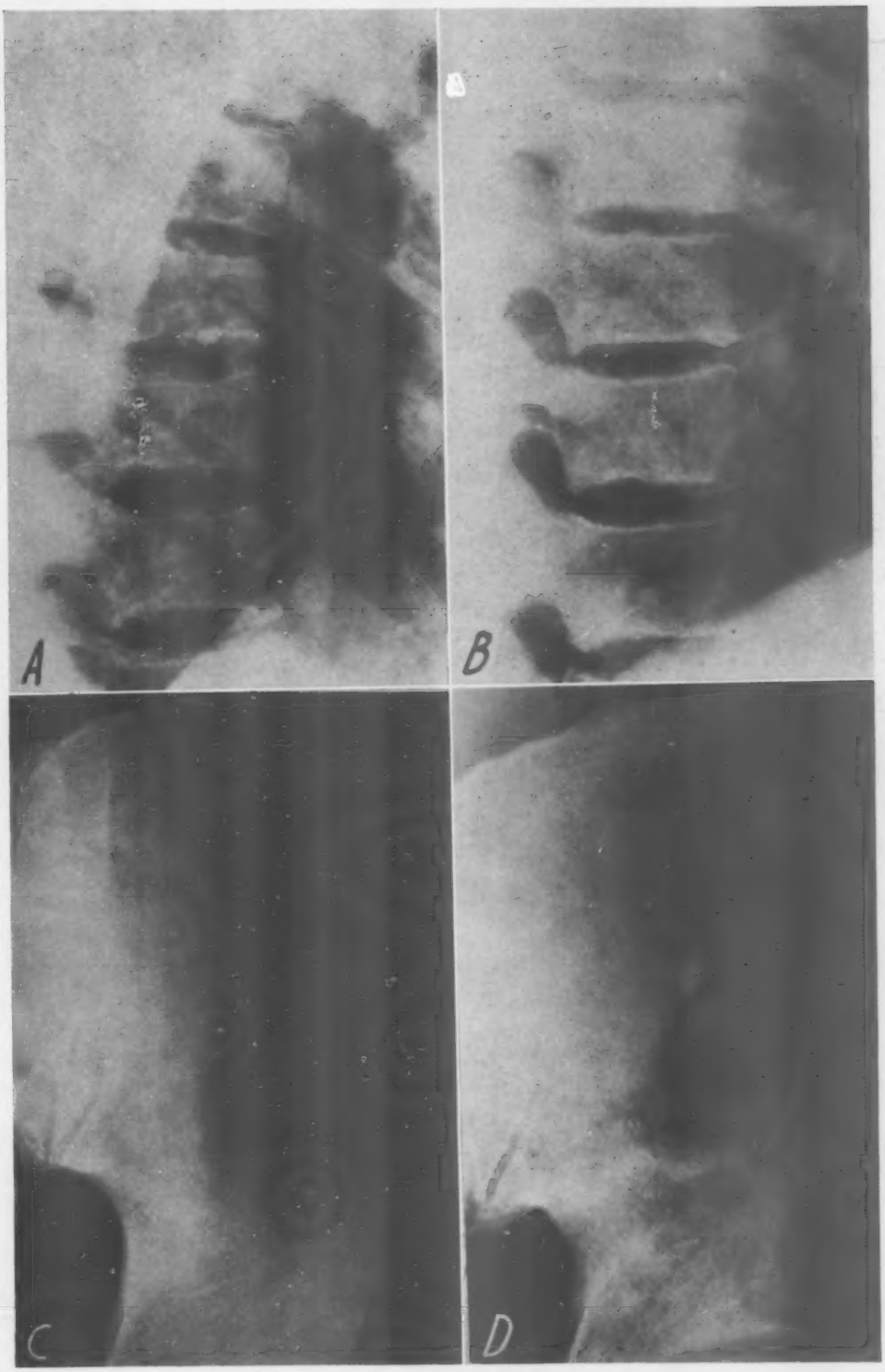


FIG. 11. Case IX. *A*, spotty sclerosis of lower dorsal vertebral bodies with coarsening of trabeculation and irregularity of structure (1945). *B*, same area one and one-half years later. Despite differences in technique, progress of sclerosis recognizable. *C*, normal appearing structure of innominate bone in 1940. *D*, same bone as in 1946, revealing fuzzy densities in lower part of the innominate bones, also in areas of the sacroiliac joint.

the endosteum. It was composed of long spindle-shaped cells. Osteoblasts and osteoclasts were absent (Fig. 12*A*, 12*B*, 12*C*).

Bone changes were almost identical in our second case (Case x) of chronic non-leukemic myelosis (Fig. 13). In this instance a male, white, American, aged fifty (E34058), came for roentgen examination because of back pains and loss of weight. Two years earlier he had been roentgenographed and diffuse density of his bones caused an erroneous diagnosis of cancer of the prostate. At physical examination a tremendously enlarged spleen and a large liver were found. The prostate showed no abnormal findings. He was fairly comfortable and worked in an industrial plant.

His blood count (August 18, 1943) was: red blood cells, 4,800,000, hemoglobin 85 per cent (Sahli), gm. 13; white blood cells 9,000, with 46 per cent polymorphonuclears (banded 40 per cent, segmented 6 per cent), 1 per cent eosinophils, 2 per cent basophils, 24 per cent myelocytes, 11 per cent metamyelocytes, 4 per cent myeloblasts, 10 per cent lymphocytes, and 2 per cent monocytes. A few nucleated blood cells were also seen.

No change had occurred in his count during the following year, after which time he felt so well that he postponed, indefinitely, any medical attention.

DISCUSSION

Clinical and hematological features were not uniform in our cases. No basic differences could be found between the symp-

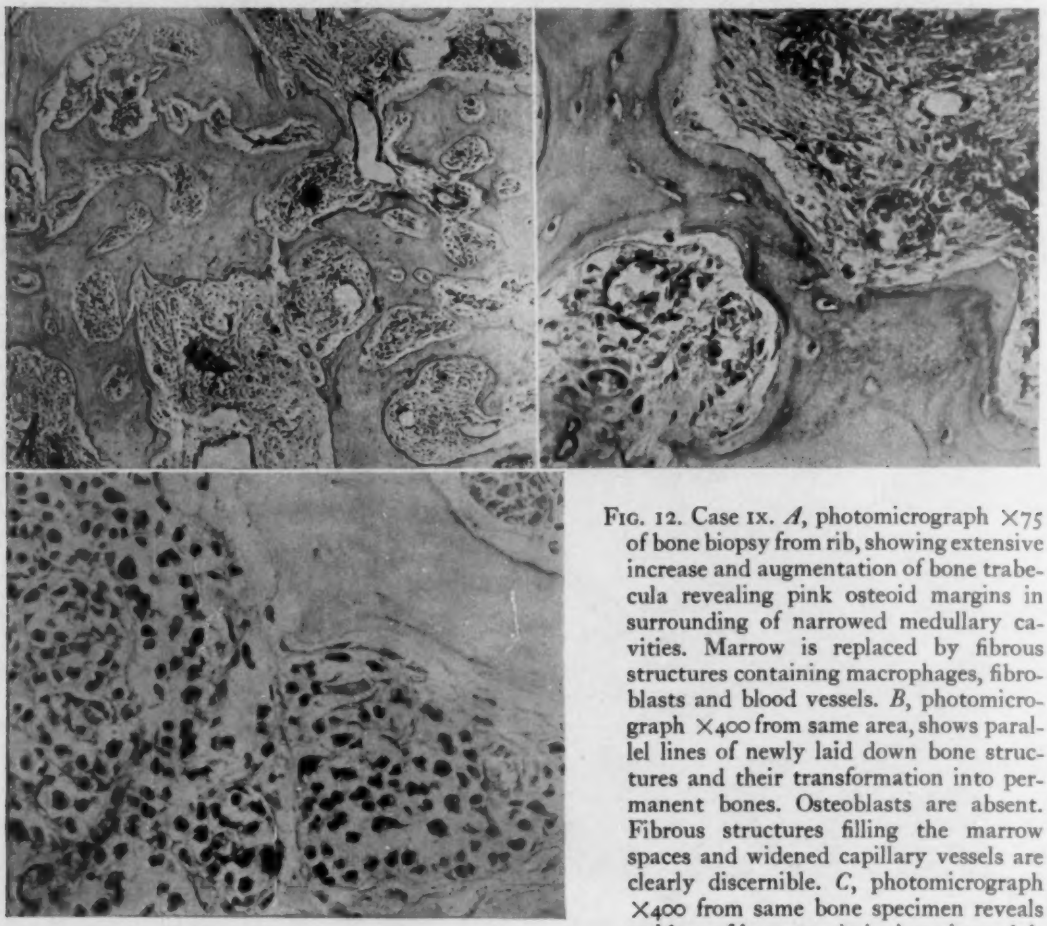


FIG. 12. Case IX. *A*, photomicrograph ×75 of bone biopsy from rib, showing extensive increase and augmentation of bone trabecula revealing pink osteoid margins in surrounding of narrowed medullary cavities. Marrow is replaced by fibrous structures containing macrophages, fibroblasts and blood vessels. *B*, photomicrograph ×400 from same area, shows parallel lines of newly laid down bone structures and their transformation into permanent bones. Osteoblasts are absent. Fibrous structures filling the marrow spaces and widened capillary vessels are clearly discernible. *C*, photomicrograph ×400 from same bone specimen reveals residues of hematopoietic tissue in medullary cavities. New formation of bone tissue is here less marked than in other areas, showing fibrous marrow. Note slight increase in reticular interstices of the marrow parenchyma.

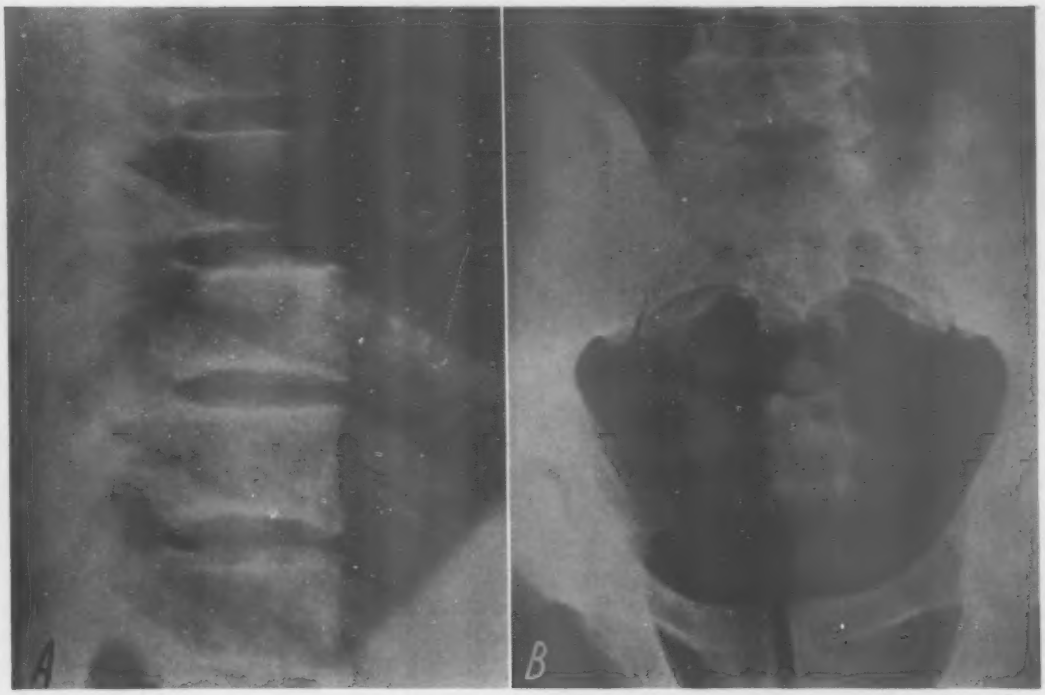


Fig. 13. Case x. *A*, osteosclerosis in non-leukemic myelosis. Roentgen appearance of lower dorsal vertebrae. Sclerosis involves bodies, processes and ribs in male, aged fifty. Splenomegaly, leukemoid blood picture. *B*, extensive density of pelvic bones and of sacrum.

tomatology and laboratory findings in cases of leukemia with bone sclerosis and those exhibiting no signs of bone formation. Prior to the roentgenological demonstration of bone sclerosis, clinical symptoms were present on the average for three months, and the patients died usually six months after signs of sclerosis were established (disregarding our cases of non-leukemic myelosis (Cases IX and X). The spleen and liver were always enlarged. The degree of anemia varied between 1.1 million and 2.4 million red blood corpuscles (with the exception of the chronic non-leukemic myeloses in which the red blood count was approximately normal). The white blood count was never very high. It ranged between aleukemic and subleukemic counts to 50,000 white cells in those cases in which sclerosis was exhibited. Specific white cells, including immature cells, ranged from 37 per cent to 99 per cent.

The roentgenological appearance of the bones varied according to localization and structure of the densities. In 2 cases it was monostotic, in 5 cases polyostotic, and in 3 cases panostotic. In the long bones the sclerosis was characterized by thickening of the cortex and, at times, dissociation of its texture. Panostotic sclerosis was more marked in non-leukemic myelosis than in our case of lymphatic leukemia. Frequently the densities were associated with periosteal proliferations. The marrow cavity was, in some instances, entirely filled by newly formed bone tissue. This was caused either by diffuse thickening of the cortex or by development of circumscribed bony proliferations.

Differential diagnostic problems were present in Case I in which the possibility of congenital syphilis had to be excluded. Case X was diagnosed prior to our studies as bone forming metastases of a cancer of the prostate.

No basic differences could be observed in the histological appearance of the marrow in different forms of sclerosis. Regard-

less of whether the leukemia was monocytic, lymphatic, or aleukemic lymphatic, bone sclerosis was always preceded by fibrous transformation of the marrow. In non-leukemic myelosis the smallness of the medullary cavity produced a different distribution of collagenous fibers than was seen in other forms of leukemia. We also had the impression that the fibers were much heavier. In the case of non-leukemic myelosis, connective tissue cells were more numerous, they were spindle shaped, and lay closer to each other than in other forms of leukemia. In other words, the fibrous tissue gave the impression of being more adult and differentiated in non-leukemic myelosis than in all other forms of leukemia. Bone formation occurred as direct metaplasia of fibrous (collagenous and reticular) interstices into woven bony lamellae.

Periosteal ossification followed the pattern of callus formation. It was preceded in our cases by minute destructions of the cortex. No elevation of the periosteum itself by leukemic infiltrates was observed.

The similarity between the microscopical and roentgenological findings in leukemia and non-leukemic myelosis convinced us that in both groups of diseases the primary change seems to be the replacement of the bone marrow by leukemic proliferations. We suppose that later on the marrow of a single or of several bones becomes depopulated of its leukemic structures. In cases of monostotic or polyostotic involvements probably a local cause has to be made responsible for the development of the sclerosis. This could be localized nutritive disturbances, obliteration of nutritive arteries, etc. Following the disappearance of parenchymal cells fibrosis develops and this is the precursor of osteosclerosis.

In panostotic sclerosis, the disappearance of active marrow occurs in most parts of the skeleton and, hand in hand with this event, a progress of bone sclerosis can be observed (Case IX). Such a progression of bone changes has already been described.[35,37]

The cause of the cellular depopulation of hematopoietic and leukemic marrow in panostotic sclerosis is unknown. The theory most favored is that a toxin acts on the marrow causing fibrosis (Donhauser[10]). Mettier and Rusk[28] believe the irritation of the hyperplastic cells themselves may cause fibrosis. Vaughan and Harrison[38] point out that all the involved cells arise from the primitive mesenchymal cells of Maximov, the proliferation of which gives the sclerosing effect that results in extramedullary hematopoiesis and the special type of blood picture.

It is reasonable to believe that in panostotic sclerosis no localized causes can be considered as inducing factors for the depopulation of the bone marrow. Observations of changes of the leukemic marrow following influences of chemicals, toxins or radiation caused previous authors to believe that such factors may destroy hemopoietic as well as leukemic cells (Klemperer[22]).

Being cognizant of the progressive fibrosis of the marrow parenchyma seen in the late stages of hemolytic anemia of infants (Caffey,[6] Kraft and Bertel[24]), we present the possibility that the toxin could be related to maternal anti-Rh agglutinins produced by iso-immunization of an Rh negative mother by Rh positive fetal erythrocytes. Kraft and Bertel[24] observed recently that bone sclerosis in a late case of sickle cell anemia was very similar to that seen in the panostotic form of sclerosis in chronic non-myeloid leukemia.

We had no opportunity to carry out such tests on our patient, but we feel that studies along these lines may prove of value and contribute to the solution of some of these unsolved problems.

SUMMARY AND CONCLUSIONS

Ten cases are presented in which sclerosis of the skeleton, or of parts of it, is associated with leukemia or non-leukemic myelosis.

The series includes three cases of monocytic, five of lymphatic leukemia, and two of non-leukemic, chronic myelosis. The roentgen appearance of the bones and the significance of roentgenological features are discussed.

In five instances roentgen findings are compared with autopsy specimens of bones of the same patient. In one case bone biopsy was used for the same purpose.

The sequel of events leading to sclerosis could be reconstructed as (1) leukemic infiltration of the bone marrow, (2) disappearance of leukemic cells (depopulation of the marrow), (3) fibrosis replacing the leukemic infiltrates, and (4) bone tissue developing in fibrous areas.

Tissue mechanism: Newly formed bone lamellae develop in fibrous marrow by direct metaplasia of fibrous interstices into woven bone structures. In cases of monocytic leukemia fibrils seem to be also formed as an exoplasmatic activity of leukemic cells. In other leukemic disorders they originate from adventitial and reticular structures.

Periosteal bone shells develop according to patterns of callus formation in the presence of multiple but minute destructions of the cortex. There is no indication in our findings of lifting of the periosteum by leukemic infiltrates.

The cause for the formation of collagenous fibers, the precursors of osteogenesis, is not evident. It is, however, reasonable to believe that in cases of monostotic and polyostotic manifestations of sclerosis, local changes like nutritive disturbances may be a cause of the depopulation of the leukemic marrow. This induces the development of collagenous fibers. In our cases of monocytic leukemia, fibers are formed by the leukemic cells themselves.

In problems of panostotic involvement the authors join in the theory of Donhauser[10] who feels that an unknown toxin may give the first impulse to the development of changes leading to sclerosis. As one approach to this problem we suggest determination of the possible presence of maternal Rh agglutinins in the blood of the patient.

Practical roentgenological results and applications of these studies are:

1. Roentgenological signs of sclerotic bone changes as presented in this paper should arouse the suspicion of leukemic disorders.

2. Monostotic and polyostotic sclerosis occurs according to our observations most frequently in monocytic leukemia and in aleukemic lymphadenosis.

3. Panostotic sclerosis (entire skeleton) is most characteristic of non-leukemic myelosis. In one of our cases it occurred in lymphatic leukemia.

4. Panostotic sclerosis accompanied by great enlargement of the spleen gives a definite diagnosis of non-leukemic, chronic myelosis.

5. Splenectomy and roentgen therapy are inadvisable in cases of panostotic sclerosis. Either of them may hasten the death of the patient. Many such patients have lived comfortably for thirty years or more without such treatment.

6. Roentgenological signs of monostotic or polyostotic sclerosis in leukemia indicate the tendency of the bone marrow to become fibrous and aplastic. However, it seems to indicate no atypical course of the leukemia.

7. Periosteal bone formation in long bones is preceded by multiple, small (often microscopic), areas of bone absorption in the cortex. At times this may become visible on the roentgenograms.

2361 Clay Street
San Francisco, Calif.

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VOL. 61, NO. 1

LUMBAR INTERVERTEBRAL DISC PROTRUSION CONTRALATERAL TO THE SIDE OF SYMPTOMS AND SIGNS*

MYELOGRAPHIC VERIFICATION IN TWO CASES

By J. P. MURPHY, M.D.

WASHINGTON, D. C.

SURGICAL opinion as to the necessity for myelography in the diagnosis and treatment of protrusion of the lumbar intervertebral disc is as widely divergent as that concerning fusion of the lumbosacral spine in this condition. Bradford and Spurling[1] stated that a correct diagnosis of herniated nucleus pulposus can be made upon the basis of historical and physical findings in 80 to 90 per cent of cases and that in 50 per cent the lesion can be localized accurately to the affected interspace. Another point of view maintains that routine myelographic examination in almost all cases not only affords objective confirmation of the diagnostic impression of disc protrusion and localizes the pathology exactly, eliminating needless exploration and shortening the operative time, but reveals the presence of multiple disc herniations. Roentgenographic examination with pantopaque enabled Echlin, Ivie and Fine[2] to diagnose bilateral protrusions in 2 cases, compression of the cauda occurring at the fourth lumbar interspace on one side and at the fifth on the other. Whitcomb[3] reported 3 more such cases of bilateral herniations at different levels in the lumbar region, again diagnosed by myelography and verified surgically.

No doubt such variants of discogenic disease are in the experience of most neurosurgeons. This report is intended only to emphasize the value of myelographic investigation of patients with obvious protrusions of the lumbar intervertebral discs in revealing the unsuspected or obscure. The clinical impression in both of the 2 cases described below was that of herniated nucleus pulposus on the left, and myelograms disclosed the protrusion to lie on the right.

REPORT OF CASES

CASE 1. E. P., a railroad fireman in his late twenties, was admitted to the Illinois Neuropsychiatric Institute on July 23, 1946, because of severe pain in the left low back, present intermittently for the preceding two years, and sciatica involving the left leg, constant for the previous several days. Examination disclosed limitation of forward bending, sciatic pain on passive straight leg raising to 45 degrees, and virtual absence of the left ankle jerk. Pain was greatly increased by coughing or sneezing. There were no complaints referable to the right leg. There was a history of injury to the low back fifteen years past, and the patient had strained himself while shovelling coal very shortly before admission.

Myelography (roentgenoscopy and spot films) was undertaken, using 3 cc. of pantopaque. Much to the surprise of the examiners, a large concave, lateral filling defect was seen on the right, the side contralateral to pain and objective findings, at the level of the fourth lumbar interspace (Fig. 1). There was no defect on the left at any point.

Under general anesthesia the spines and laminae of L-4 and S-1 were exposed bilaterally. Retraction of the nerve root to the right at the fourth interspace disclosed a thin annulus which was not protruding. Incision of the membrane resulted in the obtaining of many fragments of loose cartilage. The interspace was then explored on the right (the side of myelographic defect) and a large piece of nucleus pulposus was seen to have perforated the ruptured capsule and was displacing the nerve root and caudal sac to the left.

The patient was discharged from the hospi-

* From the Department of Neurology and Neurological Surgery, George Washington University School of Medicine, Washington, D. C.

FIG. 1. Case I. A concave filling defect is seen at the level of the fourth lumbar interspace on the right. Lateral protrusion (rupture) of the intervertebral disc on the right verified at operation.

tal two weeks after operation, asymptomatic save for slight numbness of the sole of the left foot. Three weeks later he was readmitted with recurrence of the same symptoms in the left back and leg. Reoperation failed to reveal further disc protrusions at any level. A frayed left fourth lumbar nerve root was clipped and severed, and spinal fusion performed. Symptoms gradually subsided, and the patient was again discharged without complaints.

CASE II. I. R., a thirty-eight year old white farmer, was examined on May 5, 1947, because of severe pain in the left lower leg and back for the previous five months. Four years before, the patient had been struck by heavy steel wedges on the left hip. Five months prior to examination he had been lifting heavy poles, and upon straightening suddenly felt a sharp pain in the left buttock and back. Sciatica developed rapidly, but subsided during three weeks of adequate conservative therapy. The patient then returned to work on his farm. While he was plowing, excruciating pain reappeared in the left leg and persisted to a degree sufficient to prevent further activity.

Forward bending was limited to 60 degrees from the horizontal. There was paravertebral pressure tenderness from the spines of L-3 to L-5. Straight leg raising was positive at 45 degrees from the horizontal. The patient outlined an area of hypalgesia and parasthesia on the anteromedial aspect of the left thigh, knee, and upper shin. This was confirmed objectively and corresponded roughly to the third lumbar dermatome (Foerster). Both knee and ankle jerks were equally active. There were no symptoms or signs involving the right leg.

The patient was admitted to the Doctors Hospital (Washington, D. C.). Myelography

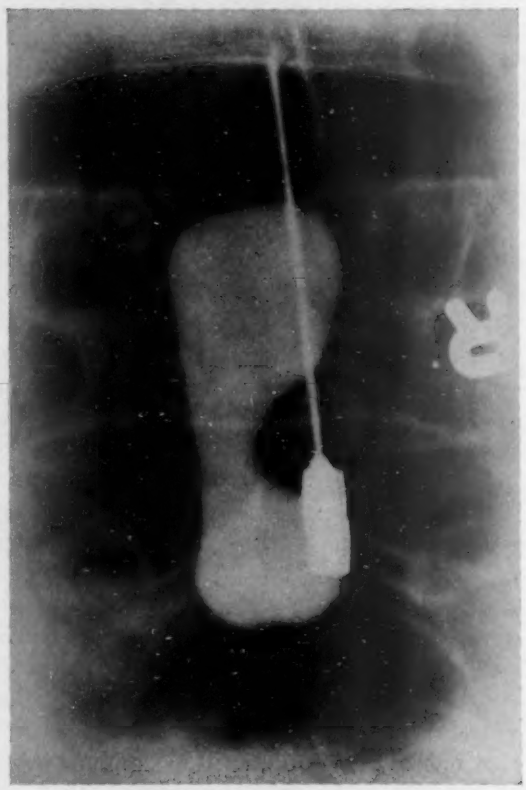

FIG. 2. Case II. A rounded, concave filling defect is seen in the pantopaque column at the fourth lumbar interspace on the right. Lateral protrusion of the disc on this side removed surgically.

(Dr. Frederick O. Coe) disclosed, instead of the expected high lumbar protrusion on the left, a consistent, concave filling defect on the right, opposite the fourth lumbar interspace (Fig. 2). The pantopaque column filled well on the left at all levels.

Lumbosacral spines and laminae were exposed bilaterally. After removal of the ligamentum flavum from the fourth interspace on the right a large protrusion of the intervertebral disc was seen. Incision of the capsule permitted the withdrawal of a sizeable fragment of loose cartilage, and much more softened disc material was removed by currettement. Exploration of the same interspace on the left (the side of symptoms) was completely negative.

Recovery of sensation on the anteromedial aspect of the left leg commenced the day of operation, and by the time of discharge (ten days postoperatively) was virtually complete. The patient declared that pain in the left testicle and groin (not mentioned during the preoperative examination) had also receded. There was never any complaint of pain in the right leg. Six weeks following discharge he remained well and asymptomatic. An island of hypalgesia was present on the inner aspect of the left knee.

COMMENT

Had operation been undertaken without myelography, the site of major pathology might not have been investigated in either of these cases. Bilateral exploration of the lumbar interspaces was performed in each, because of discrepancy between clinical and roentgenographic findings, but the disc protrusions were found only where the myelograms had indicated that they were present. Without contrast medium examination, the usual unilateral exposure might well have resulted in persistence of symptoms and signs, inasmuch as the wrong side would have been explored.

SUMMARY

Two cases of lateral protrusion of lumbar intervertebral discs on the side opposite to symptoms and signs are reported. Myelography enabled accurate exploration and removal of large herniations of the nucleus pulposus, which might have been missed had operation been undertaken upon the basis of clinical examination alone. The value of routine myelography in lumbar discogenic disease is therefore illustrated.

1911 R St., N.W.
Washington 9, D. C.

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BILATERAL POSTTRAUMATIC ULNOCUNEIFORM ARTICULATION

By HENRY MILCH, M.D.
NEW YORK, NEW YORK

UNDER normal circumstances the ulna does not articulate with the carpus. Its lower end, the head, articulates with the lesser sigmoid notch of the radius. The interposition of the triangular fibrocartilage prevents any direct contact with the wrist joint proper. The wrist joint is a condyloid joint in which the first row of carpal bones articulates laterally with the inferior surface of the radius and medially with the inferior surface of the triangular fibrocartilage. The triangular fibrocartilage is a somewhat pyramidal structure which is attached by its apex to a depression existing between the head and styloid process of the ulna. The apex is the thickest portion of the cartilage. From it the fibers fan out to form a broad, thin base which is inserted horizontally along the inferior margin of the lesser sigmoid notch of the radius. The lower concave surface articulates with the carpus, the upper concave surface with the head of the ulna.

Because of this anatomical arrangement the radius and the carpus form a functional radiocarpal unit, in which the carpus is dominated by the position of the radius. Rotation of the wrist follows rotation of the radius and takes place around an axis which runs from the superior to the inferior radioulnar joints.

FIG. 1. With both hands in pronation, there is seen radial deviation of the hands and prominence of the ulnar styloid.

This relationship is predicated upon the maintenance of normal length relationships between the two bones. If for any reason the ulna becomes shorter than normal and its pressure against the triangular cartilage is lost, ulnar deviation of the hand and finally corresponding curvature of the radius occurs. If, on the other hand, the radius should become relatively short, the carpus moves proximally. Because of the downward projection of the ulnar head, this is impossible and the carpus must subluxate anteriorly or radially to avoid impingement against the ulnar head. This situation commonly seen after inadequately reduced Colles' fracture has been improperly designated as posterior subluxation of the ulna.

In those cases in which the disproportion develops slowly so that the wrist can deviate radially in proportion to the growth of the ulna, the ulnar head may gradually make contact with the cuneiform bone and establish a new inferior joint about which the rotation of the forearm may take place. This is seen in those cases in which the lower end of the ulna is subperiosteally resected. As the new, plastic bone is formed, the carpus exerts a medial pressure and the lower end of the ulna deviates to the ulnar side. A somewhat similar situation is seen in the following case:

Rufus F., aged thirty-eight, presented himself in October, 1945, for a routine pre-employment examination. At this time the marked prominence and radial deviation of the hand commonly seen in malunited Colles' fracture was noted (Fig. 1). Because they so closely resembled the typical appearance of forward

luxation of the carpus, the wrists were examined for evidence of "so-called posterior dislocation of the ulna." The ulnar styloid was found to extend about 1 inch below the level of the radial styloid. It was noted with surprise that there was a practically normal range of flexion and extension of the wrist with no limitation of pronation and supination. Radial abduction was normal but the wrist could not be adducted to the midline. There was no dislocation of the ulna and no anteroposterior laxity. The patient had a powerful grasp.

The explanation of this peculiar situation became apparent when the roentgenogram was examined (Fig. 2). On both sides the radius was seen to have become relatively shorter than the ulna. The lower ends were broader than normal and were obviously irregular. The sigmoid cavity was notched and had clearly not articulated with the ulnar head for many years. As in normal instances, the carpal scaphoid and semilunar articulated with the under surface of the radius. On the right side the ulnar styloid was not united to the head, but otherwise both ulnar heads appeared to be relatively normal in outline. On both sides, however, the ulnar heads articulated with the medial aspects of the cuneiform bones which were correspondingly facetted. The hands were in radial deviation and the carpal bones showed what were considered to be configurational adaptations to their abnormal position. The lack of any limitation of rotation was obviously due to the fact that the ulna lay in the same plane as the radius and the carpal bones. The contact of the radiocarpal mass with the ulna had been simply displaced distally and instead of an inferior ulnoradial joint an inferior ulnocuneiform joint served as the fulcrum about which rotation occurred.

When the patient was carefully questioned about his condition, he recalled that as a child of ten he had fallen head first out of a tree. With his hands outstretched, the impact was received entirely on the radial epiphyses. At the time nothing noteworthy was seen and the patient was treated simply by a short period of splinting. However as the years passed he noticed gradually increasing prominence of the ulnar heads with deviation of the wrists toward the thumb side.

In all probability this patient suffered a compression of the inferior radial epiphyses. At the time of injury there was nothing to do and the child was simply splinted to relieve pain. As the years passed, the damage initially suffered by the epiphyseal cartilage resulted in retardation of radial growth and premature ossification of the epiphyseal line. The styloid process fractured at the time remained ununited while both ulnas continued to grow at their normal rates.

Fig. 2. Note relative shortening of both radii. The lower ends of the radii are broader than normal and somewhat irregular. On the right side the ulnar styloid is ununited. The scaphoid and semilunar bones articulate with the radius which does not make contact with the ulna at the sigmoid notch. Both ulnar heads articulate with the cuneiform which presents a well formed articular facet for their reception.

Because of the fact that ligamentous structures of the wrist were not seriously injured at the time of the injury, the ulna remained in its normal plane. As it continued to grow distally its lower end came into contact with the cuneiform and in time a facet for its reception was developed. The radial deviation of the hand was gradually accomplished by the pressure of the ulnar head, so that dislocation of the carpus did not occur, and no limitation of rotation resulted.

The patient was able to do hard manual labor without any noticeable impairment of function. The question of aesthetics did not interest him and so no suggestion of operative correction was made.

225 West 86th St.
New York 24, N. Y.

MIXED TUMORS OF THE PALATE*

LATE RESULTS IN FIVE CASES TREATED BY TELECURIETHERAPY

By MAX CUTLER, M.D.

CHICAGO, ILLINOIS

THE purpose of this report is to present the results of irradiation in 5 mixed tumors of the palate. Only 5 patients suffering from this condition were treated between 1938 and 1942. Four were treated by telecurietherapy and supplementary interstitial irradiation, and one by telecurietherapy alone. The periods of observation since treatment vary between six and nine years. The diagnosis was confirmed by biopsy in 4 cases. Four patients are well and free of disease nine years, seven years, six years and six years, and one died eight years after treatment with a recurrence that had been present for three years.

CASE REPORTS

CASE 1. N. N., male, aged forty, tumor of hard palate of eight years' duration. Examination disclosed a large ulcerated tumor measuring about 6 cm. in diameter occupying almost the entire roof of the mouth, to which it was attached by a broad base (Fig. 1). There was no cervical adenopathy. Biopsy showed a highly cellular mixed tumor of salivary gland type. Treatment consisted of telecurietherapy as follows: Radium content 10 grams, filtration 1 mm. platinum, distance 12.5 cm., portal 6 by 6 cm., 4,000 milligram-hours for twenty-five days (June 14, 1939 to July 21, 1939), total dose 100,000 mg-hr. The tumor dose was 1,875 gamma roentgens. All treatments were given through one right lateral portal 6 by 6 cm. There was excellent regression of the lesion over a period of approximately two months, leaving a small remnant which was treated by supplementary radiation in the form of removable platinum radium element needles as follows: Three needles each containing 2 mg. (2 cm. active length) and four needles each containing 1 mg. (1 cm. active length), filtration 0.5 mm. platinum. The seven needles containing a total of 10 mg. were inserted uniformly around the remnant and were removed after 144 hours, total dose 1,440 mg-hr., tumor dose 4,142 gamma roentgens. The interstitial irradiation was performed on September 14, 1939. The remnant regressed slowly and did not disappear entirely until May 11, 1940, approximately eight months after the radium insertion and eleven months after telecurietherapy. The patient is now free of disease more than eight years. There are no demonstrable radiation injuries (Fig. 2).

FIG. 1. Case 1. Extensive aberrant mixed salivary gland tumor before treatment.

FIG. 2. Case 1. After treatment.

* From the Chicago Tumor Institute. Aided by a grant from the National Cancer Institute. Presented at the Thirtieth Annual Meeting, American Radium Society, Chicago, Ill., June 20–22, 1948.

Comment. The main point of interest in this case is the apparent cure of a very extensive, so-called radioresistant mixed tumor of the palate by intensive telecurietherapy. Another interesting feature is the slow rate of regression following treatment. On several occasions it was difficult to resist the temptation to intervene surgically and only previous experience with the slow regression of lesions of this type prevented an unnecessary surgical intervention.

Case II. W. P., female, aged sixty-eight, tumor of the palate first noted about two months before admission. Biopsy had been performed and showed mixed tumor of salivary gland type. Examination made on June 18, 1939, disclosed a mass about 4 cm. in diameter with an ulcerated surface about 2 cm. in diameter situated in the soft palate slightly left of the midline. The lesion had the classical clinical appearance of a mixed tumor. Between May 1, 1939, and June 9, 1939, the lesion was treated with telecurietherapy through one left lateral portal 6 by 6 cm. with the following factors: Radium content 2 grams, filtration 1 mm. platinum, distance 10 cm., 2,000 mg-hr. daily for thirty consecutive days, total dose 60,000 mg-hr., tumor dose 1,448 gamma roentgens. Supplementary radium therapy was given between June 24, 1939, and July 1, 1939, in the form of a radium mold as follows: Radium content 35 mg., distance 7 mm., filtration 1 mm. platinum, surface area 7.8 cm.^{2}, 167 mg-hr. daily for seven days, total dose 1,170 mg-hr., tumor dose 2,633 gamma roentgens, total tumor dose 4,081 gamma roentgens. The lesion disappeared one month later. Approximately one year later, on May 31, 1940, interstitial irradiation was performed on a suspicious area of recurrence as follows: Four 2 mg. radium element needles were inserted for 144 hours, total dose 1,152 mg-hr. The patient remained free of disease until September, 1944, when she developed severe pain and on July 16, 1946, more than six years after the last radiation treatment, a definite recurrence was noted. Death occurred on November 9, 1947.

Comment. Because of the patient's poor general condition, the initial irradiation was discontinued after a tumor dose of 1,448 gamma roentgens had been administered. This dose proved inadequate to effect a cure although the patient remained free of disease for more than six years.

Case III. R. McC., male, aged fifty-nine, swelling of roof of mouth for two months. A biopsy was performed and a microscopic diagnosis of adenocarcinoma was made, but a review of the sections showed a highly cellular mixed tumor of salivary gland type. Examination disclosed a freely movable, encapsulated tumor about 5 by 4 cm. in the soft palate, right of the midline. There was a linear superficial ulcer representing the site of recent biopsy. There was no cervical adenopathy. The lesion had the classical appearance of a mixed tumor of salivary gland type. The microscopic structure was essentially adenoid cystic. Treatment consisted of telecurietherapy as follows: 3,000 mg-hr. daily to each side of face (lateral portals) for twenty-two treatment days between June 17, 1940, and July 12, 1940, radium content 10 grams, filtration 1 mm. platinum, distance 12.5 cm., portals 6 by 6 cm., total dose 96,000 mg-hr. to each portal, grand total 192,000 mg-hr., tumor dose 3,109 gamma roentgens. A pronounced "epithelite" developed over the tumor about the sixteenth day. The lesion regressed slowly and on August 31, 1940, about seven weeks after the completion of telecurietherapy, a small remannt was treated with interstitial irradiation as follows: Five platinum radium element needles each containing 2 mg. of radium (active length 2 cm.), filtration 0.5 mm. platinum were inserted for 122 hours, total dose 1,220 mg-hr., tumor dose 3,812 gamma roentgens, total tumor dose 6,921 gamma roentgens. Three months later the remnant had disappeared. The patient is well and apparently free of disease at this time, about eight years after the last radium treatment, and there are no demonstrable radiation effects except a small shallow defect about 3 mm. in diameter in the mucous membrane at the site of the original lesion.

Comment. The interesting feature in this case is the intensity of the irradiation without radionecrosis and without demonstrable radiation injuries eight years after treatment. This case represents an eight year control of a moderately advanced so-called radioresistant mixed tumor by intensive telecurietherapy supplemented by interstitial irradiation. The highly cellular

FIG. 3. Case v. Aberrant mixed salivary gland tumor before treatment.

microscopic structure places this case in Ahlbom's "semimalignant" group and originally led to a diagnosis of adenocarcinoma which was later reclassified as a cellular mixed tumor.

CASE IV. H. C., aged sixteen. On January 3, 1941, a freely movable, encapsulated tumor 2.5 cm. in diameter was enucleated from the left soft palate. Examination of the microscopic slides disclosed typical mixed tumor of salivary gland type. Examination on February 10, 1941, disclosed a local recurrence about 1.5 cm. in diameter. Treatment consisted of telecurietherapy as follows: Radium content 10 grams, filtration 1 mm. platinum, distance 12.5 cm., portal 5 by 5 cm., 10,000 mg-hr. daily for twelve treatment days given between February 10, 1941, and February 22, 1941, one left lateral portal, dose 120,000 mg-hr., tumor dose 2,820 gamma roentgens. An "epithelite" developed over the lesion about the fifteenth treatment day and a "moist epidermite" over the skin portal about the twenty-sixth day. The lesion disappeared and the patient is well and apparently free of disease six years after treatment without demonstrable signs of radiation injury.

Comment. The interesting feature in this case is the intensity of the treatment consisting of 120,000 mg-hr. in twelve consecutive treatment days without evidence of injurious radiation effects six years after treatment. This case represents a six year control of a recurrent mixed tumor of salivary gland type in a boy sixteen years of age.

CASE V. A. B., female, aged seventy-eight, growth in roof of mouth for thirty years. Examination disclosed a very extensive lesion about 7 cm. in diameter in roof of mouth with an ulcerated center about 2 cm. in diameter (Fig. 3). Treatment consisted of telecurietherapy as follows: Two grams of radium, distance 6 cm., portal 7 cm. in diameter, one hour daily for twelve consecutive days, total dose 24,000 mg-hr., tumor dose 1,233 gamma roentgens. Four months later interstitial irradiation was used as follows: Ten platinum radium element needles, each containing 1.85 mg. (filtration 0.5 mm. platinum) were inserted for 120 hours, dose 2,220 mg-hr., tumor dose 6,342 gamma roentgens, total tumor dose 7,575 gamma roentgens. The lesion regressed slowly, leaving a small fibrous mass. There is no evidence of disease six years after treatment (Fig. 4).

Comment. This is an example of an extensive mixed salivary gland tumor in a patient seventy-eight years of age controlled for six years by combined telecurietherapy and interstitial irradiation.

HISTORICAL*

Mixed tumors have been recognized for many years. The first published reports on tumors of the parotid are generally credited to C. G. Siebold[33] (1793) and J. B. Siebold[34] (1797). In 1841 A. Berard[2] published a thesis with the first attempt toward classification, and there followed a series of significant contributions, notably by Billroth[3] (1859), Bruns[6] (1859) and Virchow[37] (1863). It was soon after Minssen's comprehensive review of the subject[22] (1874) that the term "mixed tumor" became firmly established in the literature.

The first demonstration that tumors exactly resembling salivary gland tumors occurred in the palate came from Stephen Paget[27] (1886) although Robin[31] (1852) and Fonnegra[13] (1883) had already described these lesions in the palate. The French authors favored the term "adenoma" and

* In the following discussion the author has drawn freely from the excellent bibliographic review in Ahlbom's monograph.[1]

these tumors soon became the subject of numerous theses: Ott[26] (1880), Fonnegra[13] (1883), Voyer[39] (1889), Pitance[29] (1897), and more recently Nesopoulous[23] (1931) and Sapet[32] (1939). De Larabrie[8] (1890) reviewed a group of mixed tumors of the lip, mouth, and palate and applied the term "glandulae molares" thus emphasizing their glandular nature, and in 1894 Eisenmenger[10] assembled 76 mixed tumors of the palate, 5 of which he had observed personally. This author used the terms "plexiform sarcoma" and "cylindroma" commonly found in the German literature suggesting an endothelial or perithelial origin but fully recognizing their similarity with parotid tumors. Volkmann's significant contribution[38] (1895) established the endothelial conception of the origin of mixed tumors and later Gontier's monograph[15] (1904) favored the theory of an entodermal-mesenchymal origin. In 1913 Heineke[16] assembled from the literature 360 cases of mixed tumors of salivary gland and McFarland[18] in 1926 added 90 new cases. Fry[14] in 1927 published 25 cases from St. Mary's Hospital, London. In 1930 Brunschwig[7] reviewed the literature on mixed tumors of the tongue and added a case of his own.

An extensive study of mixed tumors of the palate was published by Eggers[9] in 1928 recording from the literature more than 100 cases; and in 1930 Sonnenschein[35] reviewed the literature and reported 2 additional cases. In 1931 New and Childrey[24] reported results in treatment of 48 mixed tumors of the palate seen in the Mayo Clinic between 1917 and 1930. Ahlbom's monograph[1] (1935) included 62 palatal tumors of which 22 were mixed tumors. In 1941 New and Hallberg[25] reported the end results in 76 "adenocarcinomas" of the palate of the mixed tumor type and in 1942 Martin[19] reported 103 palatal tumors of which 24 were either mixed tumors or adenocarcinomas.

FIG. 4. Case v. After treatment.

ORIGIN

The variety of terms used to designate this group of tumors reflects the controversy relating to their connective tissue or epithelial origin and regarding their benign or malignant character. Ahlbom[1] has studied this problem extensively and includes in his excellent monograph a comprehensive review of the literature. It seems that most of the French pathologists accepted an epithelial origin whereas the German school favored the mesenchymal and later

the endothelial theory. The Cohnheim theory naturally gave rise to the embryonal doctrine. The recent tendency has been to return to the original point of view of the French school, namely that these tumors arise from the ducts or alveoli of the adult salivary glands. Certainly the epithelial origin of mixed tumors is now almost uniformly accepted (Krompecher,[17] Ewing[12]). Ewing, while accepting Krompecher's interpretation of an epithelial origin, points out that not all mixed tumors can be explained in this manner and suggests that a large portion are derived from the ducts and acini of the salivary and mucous glands and adds that the theory of branchial origin must be accepted for certain cases.

The microscopic distinction between benign and cancerous mixed tumors of the salivary gland presents serious difficulties. In the early literature the writers used the terms adenoma, epithelioma, sarcoma, endothelioma, and cylindroma without consideration as to their benign or cancerous character. Broders[5] uses the general term adenocarcinoma to emphasize their potential malignancy. In the belief that, given adequate time, all salivary gland tumors ultimately become cancerous, some writers (Boeninghaus[4]) prefer to use the term carcinoma, but Ahlbom[1] objects to this designation for a group of tumors which are mostly benign and agrees with Masson[21] (1924) in recognizing an intermediate "semimalignant" group. Reuterwall at the Radiumhemmet used such a classification on the clinical material reported by Ahlbom. Martin[19] uses the term "adenoid" tumors which he subdivides into adenocarcinoma and benign mixed tumor.

CLINICAL ASPECTS

Mixed salivary gland tumors constitute between 1 and 2 per cent of all tumors and about 5 per cent of tumors of the head and neck. They are most frequent in the parotid and next most common in the palate. They are also found in the submaxillary region, floor of mouth, larynx, pharynx, nasopharynx, tongue, upper lip, cheek, and orbit. The average age is between forty and fifty years, but they can occur in the very young. Thus Wagner's case[40] of a mixed sublingual growth concerned an infant of twelve weeks, and Pallier[28] observed one case in an infant of eleven months. The incidence is somewhat higher in women (Ahlbom[1], Martin[19]). Neither heredity nor trauma appears to be an etiological factor. In many cases the tumor had been present for many years before coming under observation, the average duration in most series being about six years. There is a wide variation in the clinical behavior. In the great majority of cases there are long periods of quiescence. Thus Pailler reported an inactive period of thirty-seven years and Wood,[41] fifty-three years. In Martland's patient[20] the growth was active for two years followed by thirteen years of inactivity. In some in stances the growth is steadily progressive from the beginning.

Mixed tumors of the palate rarely cause symptoms until they reach a considerable size. Tenderness and mechanical difficulty associated with use of a dental plate often first call the patient's attention to the abnormality. Pain and bleeding are uncommon. An asymptomatic growth is not infrequently discovered by the patient's dentist. Mechanical interference with swallowing or nasal obstruction may arise when the tumor has reached considerable proportions. Large tumors may fill the mouth and extend into the antrum, nose, or nasopharynx or intracranially through the ethmoid and sphenoid bones. The anterior surface of the palate is more frequently involved than the posterior.

The clinical appearance is characteristic. The tumor is hard, round or oval, elastic, sometimes pseudofluctuating, situated usually below the surface of the mucous membrane and never exactly in the midline. The small tumors are encapsulated and freely movable under the mucous membrane; the larger ones are more fixed. Ulceration occurs late and is frequently precipitated by trauma or biopsy. As a rule, no distinction can be made between benign mixed

tumors and adenocarcinoma of the palate from clinical examination. Occasionally the more diffuse borders, early ulceration, and more rapid growth may suggest a malignant course. Rarefaction and even perforation of the bone may occur from mixed tumors of the hard palate due to pressure atrophy, a condition that is often erroneously interpreted as a sign of cancerous invasion of bone.

Upon the question of how often a benign mixed tumor of salivary gland type undergoes cancerous changes there is considerable controversy. Ahlbom contends that it is incorrect to accept a long previous history as proof that the lesion was first benign. He cites proof to the contrary by quoting examples with clinical and histopathological evidence of cancer with a long history extending back over many years (thirteen years in one case) in which microscopic examination of the original slides showed all the histopathological features of cancer.

Metastasis from mixed salivary gland tumors is infrequent and occurs late, but adenocarcinoma metastasized to regional lymph nodes in 27 per cent of Martin's cases and 29 per cent of Ahlbom's 42 mixed tumors of mouth, orbit and palate. Distant metastasis, according to Ahlbom, is estimated as 25 per cent in the entire series of cancer of salivary glands and occurs late in the course of the disease. New and Hallberg state that few of their tumors metastasized. In 13 of the 53 cases affecting the palate, cervical lymph nodes were palpable on first examination. In 4 of the 13 cases the nodes were removed and showed "adenocarcinoma" of the mixed tumor type. No mention is made of distant metastasis. Regarding systemic metastasis Martin cites the autopsy records of the Memorial Hospital containing 3 patients who died of adenocarcinoma of the palate in 2 of which there was systemic metastasis. Of 63 patients who died of mixed salivary gland tumors in Ahlbom's series 19, or 30 per cent, showed evidence of systemic metastasis. It is highly probable that distant metastasis is much more common than is generally supposed. Ahlbom estimates it as 50 per cent of those who die and 25 per cent of the entire series of cancerous tumors of salivary glands.

TREATMENT

It is generally agreed that operable mixed tumors of the palate are best treated surgically and that surgical endothermy and irradiation are valuable adjuncts. Regarding the precise value of irradiation there is some difference of opinion. Thus Martin[19] advises against its use altogether except for inoperable lesions whereas Ahlbom[1] ascribes an important role to it in operable as well as inoperable tumors. New combines radium with surgery and surgical diathermy. Most reports advocate postoperative irradiation.

In 1941 New and Hallberg[25] reported 53 adenocarcinomas (mixed tumors) of the palate of which 46 lived five or more years after treatment.* Of these 46 cases, 3 out of 7 are well after irradiation alone, 13 out of 14 after surgical diathermy combined with irradiation, and 21 out of 23 after excision and surgical diathermy (with or without irradition). Ahlbom[1] (1935), who has contributed one of the most important studies on this subject, reported 254 cases of mucous and salivary gland tumors from the Radiumhemmet (Stockholm). He omits preoperative irradiation in small, encapsulated, movable, clinically benign lesions but uses it in all other cases and especially when cancer is established or suspected. In view of the prevailing impression that preoperative irradiation complicates surgical procedures, it is interesting that Hybinnette of the Radiumhemmet (quoted by Ahlbom[1]) who has had an extensive surgical experience with these tumors, believes that preoperative irradiation helps to define the lesion and actually facilitates the operation.

Ahlbom uses irradiation alone for inoperable lesions and for operable lesions which prove to be radiosensitive. In his series one adenoma of the palate disappeared and the patient was free of disease three years and

* New and Childrey[24] had reported on group of mixed tumors of the palate in 1931 with essentially similar results.

eight months after roentgen therapy. Out of six other lesions in the "semimalignant" group, one cylindroma in the maxilloethmoidal region was sterilized by teleradium and roentgen irradiation over a period of two years and four months, one patient with "basalioma" of the hard palate was well thirteen years and five months after irradiation alone, and one fibromyxoepithelial tumor of the palate disappeared after irradiation and recurred fourteen years later. The recurrence then disappeared after a second course of irradiation. Among the 254 cases of mucous and salivary gland tumors the incidence of local recurrence was only 2.5 per cent for the benign mixed tumors and only 6 per cent when the "semimalignant" tumors are included. The author ascribes this exceptionally low incidence of recurrence to the radiation part of the treatment.

The divergent views upon the value of irradiation are due largely to the different conceptions regarding the radiosensitivity of these tumors and this leads to a discussion of the terms "radioresistant" and "radiosensitive," probably the two most confusing and misleading terms in the radiological literature. The original error which has led to this state of confusion dates back to about 1924 when microscopic studies of irradiated tissues and subsequent studies of biopsy material in relation to radiosensitivity indicated the relatively higher sensitivity of nonkeratinizing, nonsquamous carcinoma as compared with keratinizing squamous carcinoma. To these nonsquamous tumors, notably in the base of the tongue, tonsil, pharynx, etc., the term transitional cell carcinoma was applied (Ewing,[11] Quick and Cutler[30]). These studies led to the conception that squamous carcinoma was more radioresistant than nonsquamous carcinoma and, generally speaking, this holds true today. From these observations many pathologists drifted into the habit of designating any tissue which showed squamous features, microscopically, as being radioresistant so that the pathological report "squamous carcinoma, radioresistant" became routine in many laboratories. The writer confesses to being among the first to commit this error. The next source of confusion developed when clinicians, notably surgeons, began to interpret a microscopic biopsy report of "radioresistance" as indicating that the lesion is incurable by irradiation and hence should be treated surgically, and curiously enough, this view is still held by most physicians. Actually, this conception is erroneous for the following reasons: (1) The biopsy is frequently not representative of the lesion. When a tumor is composed of both squamous and nonsquamous elements, it is often radiosensitive and sometimes highly radiosensitive and radiocurable and since the biopsy frequently shows a preponderance of squamous elements, the report that the lesion is radioresistant is obviously erroneous. (2) When a squamous carcinoma is not infiltrating or only slightly so, as for example on the skin, mucous membrane of lip, mouth, or larynx, it is curable by adequate and correct irradiation in spite of the presence of squamous features in the biopsy. (3) The most important source of error is the failure to take into account the advances in radiation therapy over the years which have resulted in a better understanding of the principles and a marked increase in doses and intensity of radiation. (4) An infiltrating squamous carcinoma, incurable by external irradiation alone, may be curable by combined external and interstitial irradiation, hence a biopsy report of "radioresistance" is misleading under these conditions. (5) Anyone who has observed an extensive clinical material under radiation therapy realizes the hazard of predicting radiosensitivity in any given case, because of the numerous exceptions. Thus lesions, even melanoma which is among the most "radioresistant" tumors, may disappear completely under radiotherapy.

From this discussion it is obvious that the terms radiosensitive and radioresistant should be qualified, and the suggestion is made that they be changed to "relatively radioresistant" and "relatively radiosensi-

tive" in order to help clarify the present misunderstanding. For a comprehensive review of this problem the reader is referred to Stewart's[36] excellent article on the subject.

SUMMARY AND CONCLUSIONS

Five cases of mixed tumors of the palate were treated between 1938 and 1942. All received telecuriethérapy as the essential treatment. In four cases small remnants were treated with supplementary interstitial irradiation. The disease was early in one case, moderately advanced in two and very advanced (probably inoperable) in two. One elderly patient developed local recurrence five years after inadequate irradiation* and died with disease eight years after the original treatment. The other four are living and apparently free of disease six years, six years, seven years, and eight years respectively.

Although, generally speaking, mixed tumors of mucous and salivary glands are comparatively radioresistant, there are many exceptions; furthermore the greater effectiveness of present-day intensive irradiation naturally brings within the radiocurable sphere more and more tumors formerly designated as radioresistant. The results herein reported constitute an example of this change and illustrate the fallacy of designating these tumors as being radioresistant and hence incurable by irradiation.

The excellent results of surgery and electrosurgery for mixed tumors of the palate make these procedures the methods of choice for clearly operable and benign lesions. Postoperative irradiation is an important adjunct and its use should be carefully considered in all cases and administered when clinical or histopathological evidence points to the likelihood that the operation will be followed by local recurrence. The use of preoperative irradiation should be considered in selected cases, especially in advanced lesions of borderline operability or when the age or general condition of the patient makes it preferable to avoid surgery.

The greater effectiveness of more radical irradiation makes it necessary to re-evaluate the entire problem of treatment, and since an adequate number of these tumors has not yet been treated by current techniques, an evaluation of the status of irradiation must await the late results of present methods. Since cures have been accomplished in numerous advanced cases irradiated on a palliative basis with what is now regarded as inadequate treatment, it is reasonable to expect a higher precentage of cures by more radical irradiation. These results illustrate this point. As in the treatment of all tumors each case constitutes an individual problem and the selection of the best treatment can only be made after a careful study of each patient. As regards the type of radiation, the most essential factor is that the total dose and intensity should be as large as possible without causing permanent damage to normal structures. As experience with intensive irradiation accumulates, the chances of avoiding serious complications improve and the method becomes more safe. Ahlbom points out that telecuriethérapy may have the advantage of causing less injury to the normal tissues than is caused by roentgen therapy. Our experience during the last eight years with a 10 gram radium bomb† tends to support this view.

No definite conclusions can be drawn from this small group of cases. Attention is called to the possibilities of cure without radionecrosis of so-called radioresistant mixed tumors of the palate by telecuriethérapy.

430 N. Michigan Ave.
Chicago, Ill.

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* The age and infirmity of the patient did not permit adequate treatment.

† Grateful acknowledgment is made to the Union Miniere du Haut Katanga for making available the 10 gram radium bomb.

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THE EFFECT OF HYALURONIDASE ON THE ABSORPTION OF A SUBCUTANEOUSLY DEPOSITED RADIOPAQUE SUBSTANCE*

By NORMAN SIMON, M.D.,† *and* LESTER NARINS, M.D.‡

The Mount Sinai Hospital

NEW YORK, NEW YORK

SINCE the discovery of the spreading factor by Duran-Reynals[1] in 1929, and its subsequent identification as hyaluronidase by Meyer, Dubos and Smyth[7] in 1936, many investigations on the biologic action of this enzyme have been conducted. Hyaluronidase activity in the skin can be clearly demonstrated by the intradermal spread of india ink,[4] certain dyes, and infections.[6] The enzymatic activity in the subcutaneous tissues, however, is less readily demonstrated. This report is concerned with the presentation of a roentgenographic method of demonstrating the absorption-hastening properties of subcutaneously injected hyaluronidase.

Favilli[2] and Sannella[10] have studied the effect of hyaluronidase on the subcutaneous absorption of india ink or saline solutions in experimental animals. More recently, Hechter, Dopkeen and Yudell[3] have demonstrated the latter effect in human infants. In these experiments estimations of rate of absorption were based upon the consistency to palpation of the injected area, the time necessary for the injection of a given quantity of fluid, and the time of disappearance of the swelling made by the injected solution.

In order to establish a more objective estimate of this absorption-hastening factor of hyaluronidase in subcutaneous tissue we have adopted a roentgenographic technique which lends itself to appraisal as readily as does the spreading of india ink in the skin. Radiopaque contrast media were injected into the subcutaneous tissues of guinea pigs, and serial roentgenograms were made to determine the time of roentgen disappearance of these substances. The time of disappearance in control animals was compared with the disappearance in animals in which the subcutaneous sites had previously been prepared with hyaluronidase. The hyaluronidase used in this study was an extract of bull testis supplied in a powder form in the strength of 50 TRU per mg.*

METHOD

Guinea pigs were tied in the supine position, and subcutaneous injections were made in the ventral body wall. The sites selected were right and left upper chest, and right and left inguinal regions. Control sites were injected with 1.0 cc. of normal saline, while test sites were injected with 12 or 50 TRU of hyaluronidase dissolved in 1.0 cc. of normal saline. Control and test sites were rotated in a clockwise manner in successive animals. Twenty minutes after preparation of the sites as above, all of these areas were reinjected with 1.0 cc. of a radiopaque contrast substance (neo-iopax, Schering, a 50 per cent solution of disodium N-methyl-3,5-diiodo-chelidamate). The twenty minute interval between preparation of the site with hyaluronidase and injection of neo-iopax is essential. When mixtures of hyaluronidase and neo-

* TRU=Turbidity Reduction Unit. One TRU is defined as the amount of enzyme per ml. which will reduce the turbidity of the substrate-protein complex from that obtained with 0.2 mg. to that obtained with 0.1 mg. substrate. The substrate-protein complex consists of a salt of hyaluronic acid combined with horse serum.[5]

* The hyaluronidase and neo-iopax were supplied by the Schering Corporation, Bloomfield, N. J.

† Dazian Fellow in Radiology, Service of Dr. M. L. Sussman, The Mount Sinai Hospital, New York.

‡ Resident in Urology, Service of Dr. G. D. Oppenheimer, The Mount Sinai Hospital, New York.

iopax are injected there is no absorption-hastening effect. Similar results were obtained when mixtures of hyaluronidase and diodrast were used (diodrast, Winthrop, 35 per cent solution of diiodo-pyridone acetic acid diethanolamine). Serial ventrodorsal roentgenograms were then made at ten to twenty minute intervals to record the presence or absence of radiopaque substances. Each roentgenogram usually included three simultaneously tested guinea pigs on the same 14 by 17 inch film (Fig. 1). The time of disappearance of the density due to neo-iopax was considered to be the end point (Fig. 2).

RESULTS

Ten guinea pigs were injected in a total of twenty-four sites, using either two, three or four areas in an individual animal. Of these twenty-four sites fourteen were controls, while ten were test sites. The test areas were prepared with either 12 or 50 TRU of hyaluronidase. The mean time of disappearance of neo-iopax from control sites was 113 minutes (range; 75 to 180 minutes), while the mean time of disappearance from test sites was 79 minutes (range; 20 to 120 minutes). The standard error of the mean in controls was 7.3 minutes, and in the test group 10.0 minutes.

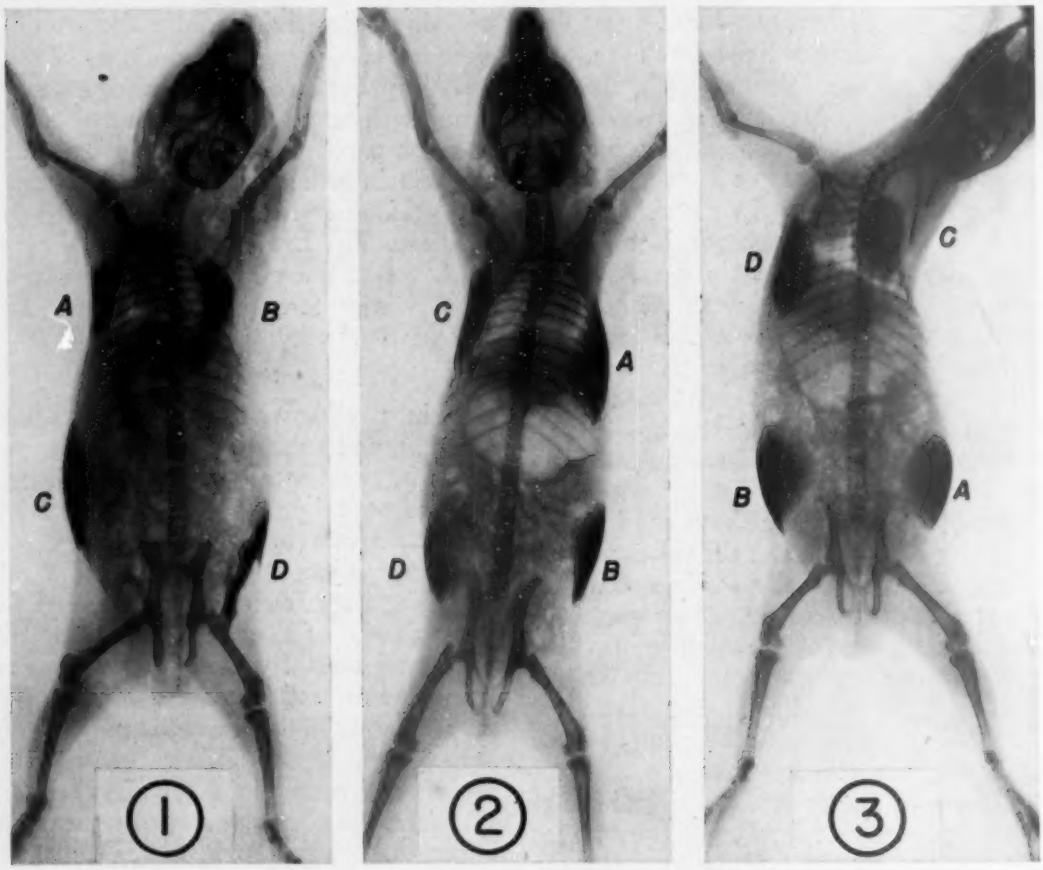

FIG. 1. Guinea pigs injected with neo-iopax in four subcutaneous sites. Preparation of each site is indicated by letters A, B, C and D adjacent to subcutaneous deposits of neo-iopax. A and D indicate controls (1 cc. of normal saline before injection of neo-iopax). Test sites labeled B were prepared with 50 TRU of hyaluronidase, and sites labeled C were prepared with 12 TRU of hyaluronidase before injection of neo-iopax. Test and control sites were rotated in successive animals as indicated.

The ratio of the difference between the mean disappearance time in test and control sites to the standard error of the difference was 2.7. This ratio indicates that the difference time is due to hyaluronidase, since the probability that it is due to random variation is less than 0.01.[8] There was no significant difference between the absorption-hastening effect of 12 as compared with 50 TRU.

FIG. 2, *a* and *b*. Showing a guinea pig immediately after injection of subcutaneous sites with neo-iopax (*a*) and thirty minutes later (*b*). Radiopacity persists in control sites A and D, while test sites B and C show almost complete disappearance of density. Note urinary bladder density in *1b*.

DISCUSSION

Hyaluronidase activity in the skin can be demonstrated by its spreading effect on intradermally injected india ink. In vitro the enzyme activity can be measured by such chemical tests as turbidity reduction,[5] mucin clot prevention, and reduction in viscosity. The present study has yielded a roentgenographic method of demonstrating hyaluronidase activity in subcutaneous tissue. This demonstration of the absorption-hastening effect of hyaluronidase uses complete disappearance of roentgen density as a definite end point. In preliminary experiments it was also found that the urinary bladder was visualized sooner in those animals in which hyaluronidase had been used to prepare the injection sites. This would seem to indicate that blood stream absorption of subcutaneously deposited substances occurs more rapidly as the result of hyaluronidase. In addition to demonstrat-

ing hyaluronidase activity in the subcutaneous tissue, the method may be applicable in the general problem of influencing absorption from subcutaneous tissues by various chemical and physical agents.

The absorption-hastening effect of hyaluronidase may also find use in subcutaneous excretory urography in infants.[9] This may make excretory urography in infants an office procedure, rather than one requiring specialized pediatric intravenous technique.

SUMMARY

1. A roentgenographic method of demonstrating the effect of hyaluronidase on the absorption of a radiopaque substance from subcutaneous tissues is described.

2. Hyaluronidase hastens absorption of a water soluble radiopaque substance from the subcutaneous tissues.

The Mount Sinai Hospital
1 East 100th St.
New York 29, N. Y.

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EFFECT OF ROENTGEN RAYS UPON *BARTONELLA*-INFECTED AND NORMAL RATS*

By DOROTHY NEUHOF NAIMAN, Ph.D.†
Department of Physiology, Health and Hygiene, Hunter College of the City of New York
NEW YORK, NEW YORK

THE study reported here was preliminary to a more extensive inquiry into the effect of roentgen rays upon the course of a rat trypanosome infection. Essentially it was desired in these preliminary experiments to determine the tolerance to roentgen rays and ability to survive following irradiation of rats of different ages. However, because the stock Sherman strain animals available were frequently infected with the common rat parasite, *Bartonella muris*, it was deemed advisable to determine first whether *B. muris* infection affects the resistance of rats to roentgen rays. This was important since roentgen rays are known to have a particularly deleterious effect upon lymphoid tissue and since splenectomy enhances the virulence of *Bartonella* infection.

Accordingly, 25 rats were given single continuous exposures of from 300 to 1,000 r over the entire body; 12 of these rats were *Bartonella*-free; the other 13 were infected with *B. muris* either several days before irradiation or four hours thereafter. The physical factors used were as follows: 200 kv., 25 ma., 1 mm. Al+0.15 mm. Cu filtration, 50 cm. target skin distance. During irradiation, the animals were immobilized in lightweight cardboard boxes. Following exposure, red and white blood corpuscle counts were made and rat weights recorded, at intervals of one to two days, until death or for twenty-one days.

In all the rats, there was an initial decrease in weight and an immediate sharp reduction in the number of circulating leucocytes. Between the seventh and twelfth post-irradiation days, those rats which still survived suffered a marked drop in circulating erythrocytes. This effect on the red blood cell count was in contrast to the results of Lawrence and Lawrence[1] who noted no significant change in the erythrocyte counts of eighty-day old rats exposed to 500 r. Some of the animals which died within a week of irradiation were emaciated, had mucous diarrhea, occasionally blood-stained, and edematous eyelids. Most frequent gross postmortem findings were petechial hemorrhages in the lungs, gross hemorrhage in the stomach and atrophy of the spleen. Table 1 gives pertinent data on the red and white blood cell counts and survival time following irradiation of the two groups of rats. (The approximate normal level of erythrocytes of these rats was 8.5 million per cu.mm., and of leucocytes, 8 thousand per cu.mm.)

No definite conclusions as to the effect of *Bartonella* infection on survival time following roentgen irradiation can be drawn from the data given in Table 1. However, it would appear that *Bartonella*-infected rats are probably slightly less resistant than are normal rats to the effects of roentgen rays. It will also be noted that the larger rats survived after exposure to 300 r and 500 r for longer periods than the smaller ones exposed to 300 r and 400 r. Since the larger animals received proportionally higher dosage because of more scatter and greater absorption, it is evident that older rats are capable of sustaining

* The work described in this paper was performed in the Department of Bacteriology, College of Physicians and Surgeons, Columbia University, and the Department of Radiotherapy, Presbyterian Hospital, New York.

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TABLE I

EFFECT OF ROENTGEN IRRADIATION OF *Bartonella*-FREE AND *Bartonella*-INFECTED RATS

Series	Number of rats	Initial weight (grams)	*Bartonella muris* infection	Roentgen irradiation (r)	Average minimum erythrocytes (10^6 per cu.mm.)	Average minimum leucocytes (per cu.mm.)	Average survival (days)
A I[1]	1	80–140	−	300	3.2	750	21+
	2		+	300	2.2	250	21+
	2		−	500	3.0	425	18+
	2		+	500	2.3	450	20.5
	2		−	750	[3]	675	6
	2		+	750	[3]	450	4.5
	1		−	1000	[3]	250	4
	2		+	1000	[3]	150	4
A II[2]	4	36–40	−	300	3.0	560	12.5
	2		+	300	2.7[4]	550[4]	10.5
	2		−	400	[3]	[5]	4
	3		+	400	[3]	[5]	4
	12	36–140	−	+	3.1[6]	532	
	13		+	+	2.4[6]	370	

[1] Series A I: rats infected with *B. muris* before irradiation.
[2] Series A II: rats infected with *B. muris* four hours after irradiation.
[3] Rats died before fall in circulating erythrocytes occurred.
[4] Results from 1 rat only; other rat had gangrenous tail.
[5] Sufficient blood for accurate count could not be obtained.
[6] Results from rats which survived long enough for fall in erythrocytes to occur.

much larger doses of roentgen rays. Similar results were reported by Quastler[3] who found that heavier mice are less sensitive to roentgen rays than lighter mice.

Since the magnitude of the irradiation effect would depend upon the age of the animal, it was clear that absolute roentgen-ray dosage could not be used in studying the effects of irradiation on infected rats of different ages. Therefore, in order to standardize the experimental conditions, an attempt was made to determine the maximum dose which would permit survival of normal (*Bartonella*-free) rats of different ages, for twenty-one days. Forty-four rats were irradiated and weighed on alternate days following exposure (Table II).

From the results given in Table II, it is evident that the maximum roentgen-ray exposure which would uniformly permit survival for at least twenty-one days in reasonably good nutrition of rats fifteen, twenty-five, thirty-five and sixty days old at the time of irradiation was, respectively, 300, 400, 500 and 500 r. It may be mentioned that these values, at least for the oldest group, are somewhat higher than those found by Leach and Sugiura[2] who reported that all 7 of their two and one-half to three and one-half month old rats that were exposed to 500 r died within eighteen days, the shortest time interval between irradiation and death being two days.

Hunter College of the City of New York
695 Park Ave.
New York 21, N. Y.

TABLE II

EFFECT OF ROENTGEN IRRADIATION ON SURVIVAL TIME OF RATS

Number of rats	Age when irradiated (days)	Roentgen irradiation (r)	Average number days before resumption of normal growth (wt.) rate	Average survival (days)
2	60	750	—	6
6		500	10	21+
2		400	9	21+
2	35	600	—	10
4		500	11.2	21+
2		350	5	21+
3		300	3	21+
2	25	500	—	21+[1]
5		400	5.4	21+
2		300	4	21+
2		250	3	21+
2		200	2	21+
2	15	350	—	14.5
5		300	4.4	21+
1		200	3	21+
2		150	1	21+

[1] Although these 2 rats survived twenty-one days, their nutrition was obviously seriously affected; four weeks after irradiation they had attained only slightly more than half the weight of their unirradiated litter mates.

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ON THE ASSOCIATION OF CERTAIN HEMATOMAS WITH MYELENCEPHALIC BLEBS IN THE HOUSE MOUSE*

By ARNOLD B. GROBMAN, Ph.D.

University of Florida

GAINESVILLE, FLORIDA

CERTAIN liver and testis hematomas and a case of *situs inversus viscerum* have been described by Bagg, in a series of papers, along with the kidney, eye, and foot abnormalities which are generally considered to be the expression of the gene for myelencephalic blebs. Apparently Bagg believed the liver and testis hematomas to be associated with the gene *my* for he wrote.[2]

However, as described above, disturbances have been noted, apparently mainly blood vascular in nature, in the tissues adjacent to the kidneys, and several instances have been recorded of similar disturbances, especially in the liver and testis, which are similar to the characteristic eye and foot lesions associated with blindness and club feet, respectively. These observations lead the writer to conclude that some common etiological relationship probably exists between the various abnormalities.

Grüneberg[4] discounts the association of *situs inversus viscerum* with the other anomalies but accepts Bagg's allocation of the hematomas. In the present note this latter allocation will be questioned.

The pertinent data from Bagg may be brought together as follows (Table I).

Somewhat comparable data have been obtained in this laboratory, from mice quite unrelated to Bagg's, and are summarized in Table II.

The liver and testis hematomas are visible externally in newborn mice while the pancreas hematomas and *situs inversus viscerum* can be determined by dissection only. Herein we shall be concerned with the former two anomalies only, since routine autopsies of newborn mice were not part of our experiment.

The mice with which Bagg worked were all descended from a single stock which had been exposed to roentgen radiation.[5] Grüneberg[4] is of the opinion that the original stock was already heterallelic for *my*.

TABLE I

Abnormality	No. Examined	No. Abnormal	Per Cent Abnormal	Source
Kidney defects	5600	1057	18.9	1929: 213
Eye defects	447	374	83.7	1923: 981 (Little and Bagg)
Liver hematomas	1817	7	0.4	1925: 287
Testis hematomas†	5600	15	0.3	1929: 213
Pancreas hematoma	A single case observed			1925: 288
Situs inversus viscerum	A single case observed			1924: 138 1925: 282, 297

† Bagg wrote:[3] "In one instance an animal had a hemorrhagic testis that was observed through the body wall soon after birth, and in later life it was found to have that testis completely missing." We have corroborated this observation to a certain extent with 2 newborn mice in which hematomas were visible through the body wall. These mice were autopsied at forty-two and forty-five days of age and in each the involved testis was considerably reduced (about 50 per cent from its fellow in linear dimension). Each contained motile sperm in its vas, however.

* The new data reported here were obtained incidental to work carried on for the Manhattan Project at the University of Rochester under contract number W-7401-eng-49.

Table II

Abnormality	No. Examined	No. Abnormal	Per Cent Abnormal	Origin of Mice	Method of Observation
Kidney defects	11333	10	0.1	Little *dba*×C57 Blk	Autopsy of adult ♂♂ and ♀♀
Kidney defects	164	0	0.0	Swiss×Bagg albinos	Autopsy of adult ♂♂
Eye defects	11333	12	0.1	Little *dba*×C57 Blk	Autopsy of adult ♂♂ and ♀♀
Eye defects	164	0	0.0	Swiss×Bagg albinos	Autopsy of adult ♂♂
Liver hematomas	19514	22	0.1	(Little *dba*×C57 Blk) ×(Swiss-Bagg albino)	External examination of newborn mice, ♂♂ and ♀♀
Testis hematomas	19514	38	0.2	(Little *dba*×C57 Blk) ×(Swiss-Bagg albino)	External examination of newborn mice, ♂♂ and ♀♀
Pancreas hematomas	>100	0	—	(Little *dba*×C57 Blk) ×(Swiss-Bagg albino)	Autopsy of newborn mice, ♂♂ and ♀♀

After the anomalies appeared the mice were selectively inbred for the defects and undoubtedly they were made homozygous for *my*.

Our material is thought to be free of *my*. Our mice are unrelated to Bagg's *my/my* mice; they originated from well-known stocks in which *my* has never been reported; and they have practically none of the kidney and eye defects associated with the gene (compare Tables I and II).

Comparison of the frequencies of testis hematomas in Table I and Table II gives a chi-square of 1.10 with one degree of freedom (P ca. 0.25), and suggests a low occurrence of testis hematomas in mice *regardless* of the presence of the gene for myelencephalic blebs.

However, the comparable values for the liver hematomas do not support a similar conclusion. Here the chi-square value of 9.09 with one degree of freedom ($P < .01$) is consistent with an association of these hematomas with *my*.

Since our +/*my* mice exhibit frequencies of the testis hematomas similar to those of Bagg's *my/my* mice, it appears likely that testis hematomas (and probably pancreas hematomas and *situs inversus viscerum*) are not always to be attributed (either directly or indirectly) to the gene *my*. The data do not support such a conclusion regarding liver hematomas although it is not unlikely that strain differences, independent of *my*, may be chiefly involved.

Department of Biology
University of Florida
Gainesville, Florida

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DEPARTMENT OF TECHNIQUE

Department Editor: ROBERT B. TAFT, M.D., B.S., M.A., 103 Rutledge Ave. Charleston, S. C.

A NEW PROCESSING ROOM ACCESSORY: THE "CLIP DRIP" TRAY*

By STANLEY H. MACHT, M.D.
BALTIMORE, MARYLAND

NO single problem is so commonly and universally encountered as that of how to best handle wet films brought from the processing room for emergency viewings. The problem is greater in the larger hospitals where the intern, the assistant resident, and the resident of a service, each in turn and at separate intervals is apt to arrive at the roentgen department and express his desire to review the "wet" films. Processing solutions, water, or combinations of both, drip from the film as they are carried from the dark room to the roentgenologist's viewing boxes. The floors are dripped upon, the viewing box shelf and any films thereon are dripped upon, and the roentgenologist's clothes are dripped upon—in fact everything but the drip cloth which is supposed to be held under the wet films bears the tell tale stains of this roentgenological industrial hazard. It occurred to the author that the solution to the problem would be a tray that would "clip" the drip. After considering various materials, "Lucite" was selected because of: (1) its resistance to water and developing chemicals; (2) its tensile strength and non-breakable qualities; (3) its light weight.

A tray was then designed and early this year one of the larger manufacturers (DuPont) fabricated samples (Fig. 1). Several trays are kept in the processing room. When a wet film is requested, a tray is snapped onto the lower bar of the film hanger (Fig. 2). The hanger may now be

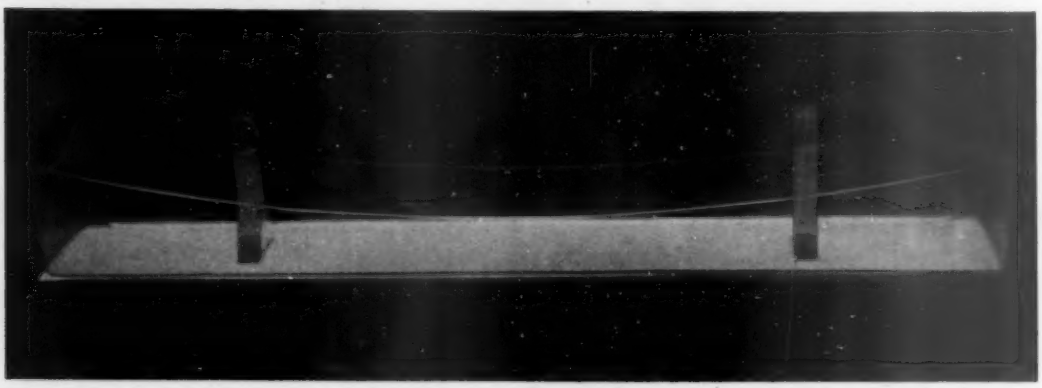

FIG. 1

*Patent pending.

lifted, tilted, or swung about. The dripping solutions are caught in the tray. When the film is held in front of an illuminator the tray holds the bottom out far enough so that the wet film does not touch the glass. The tray has been tested under actual working conditions in the Department of Radiology of a large city hospital. It has proved completely satisfactory and is therefore being described here. It has proved of special value in carrying wet films into the operating room during orthopedic operations.

Fig. 2

SUMMARY

A tray has been designed and fabricated for the purpose of catching dripping solutions from "wet films."

2307 Crest Road
Baltimore, Maryland

TRIFOCAL GLASSES AS AN AID TO THE OLDER ROENTGENOLOGIST

By RAMSAY SPILLMAN, M.D.
NEW YORK, NEW YORK

AT about the age of forty-five, the eye of the average human being has acquired a focal length that is a little longer than his arm. Among the many inventions of Benjamin Franklin, the bifocal spectacle was no mean achievement. Some persons have more trouble adapting to it than others.

In the early fifties, presbyopia has normally increased to the degree that an

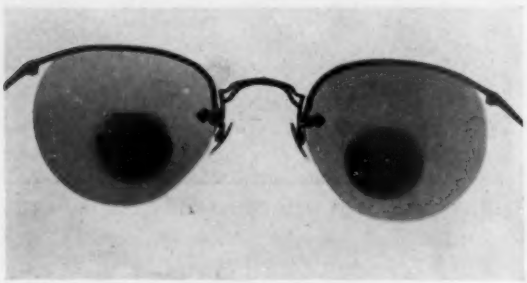

FIG. 1

adequate addition for reading differs so greatly from the correction for distance that there is an intermediate zone in which sharp vision is not obtained. This zone includes the distance at which one would normally read roentgenograms.

For persons who can accept them, trifocal glasses bridge this gap and give continuous vision. The only reason for this communication is that I find comparatively few of my colleagues in the age group to benefit from them have ever heard of them. The principle is simplicity itself, though the optical details inspire appreciative admiration. The lens is ground to uniform curvature, the difference in the strength being inherent in the different indices of refraction of the three kinds of glass involved. The accompanying roentgenogram (Fig. 1) illustrates the principle and also shows the pattern formed by the three elements. The intermediate rectangle constitutes an addition that is half the strength of the lower addition. I have found that the proper inclination of the head to throw the vision through the needed correction very quickly becomes a reflex action and involves no conscious effort; and this, notwithstanding a complicated optical prescription involving spheres, cylinders, and prisms.

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Janeway Lecture Committee: Douglas Quick, Chairman, New York, N. Y., G. Failla, New York, N. Y., F. W. O'Brien, Boston, Mass.

Representatives on American Board of Radiology: Douglas Quick, New York, N. Y., B. P. Widmann, Philadelphia, Pa., F. W. O'Brien, Boston, Mass.

Committee on Arrangements: E. P. Pendergrass, Chairman, Philadelphia, Pa., John F. Hynes, Wilmington, Del., P. C. Swenson, Philadelphia, Pa.

Advisory Committee on X-ray and Radium Protection of the National Committee on Radiation Protection: Edith H. Quimby, New York, N. Y., J. E. Wirth, Baltimore, Md.

Exhibit Committee: Robert E. Fricke, Chairman, Rochester, Minn., William Harris, New York, N. Y., Milton Friedman, New York, N. Y.

Thirty-first Annual Meeting: Ambassador Hotel, Atlantic City, N. J., June 5–7, 1949.

EDITORIALS

NITROGEN MUSTARD THERAPY

SHORTLY after World War I a few articles[1,2] appeared in the medical literature pointing to the fact that the mustard gas used in chemical warfare exerts, among other things, a toxic effect on the hemopoietic system leading to leukopenia. During World War II research carried out on a series of mustards revealed that most of these compounds also produce a cytotoxic action on the elements of the lymphoid system and to a certain extent on the proliferative processes of malignant neoplasms. This action in many ways resembles that observed following roentgen irradiation. Hence, several investigators turned their attention to the possible therapeutic applicability of some of the mustards.

Gilman and Philips,[3] in 1946, gave the first account of such applicability. These authors reviewed considerable unpublished work carried out in conjunction with the war effort and analyzed the relation existing between the chemical constitution and the biological action of numerous nitrogen mustards. They arrived at the conclusion that there is an almost infinite variety of mustards which may be prepared and that considering the great selectivity of action of some of these drugs "a measure of optimism is lent to the anticipation of future investigations." Bearing out this prediction of Gilman and Philips, during the next two years a relatively large number of articles were published in rapid succession.

It may not be out of place, therefore, to try to analyze some of the progress made and to consider the present status of the medical usefulness of the mustards.

The clinical and pathologic effects were studied in extenso recently by Graef, Karnofsky, Jager, Krichesky and Smith.[4] One sulfur and four nitrogen compounds were investigated which by using the official War Department symbols for brevity were designated, as H (bis-β-chloroethylsulfide), HN_1 (ethyl-bis-β-chloroethylamine), HN_2 (methyl-bis-β-chloroethylamine), HN_3 (tris-β-chloroethyl-amine) and TL_{301} (iso propyl-bis-β-chloroethylamine). Of the five preparations HN_2 is the most commonly used in clinical therapy and is thus of particular interest as far as this review is concerned.

Graef and his colleagues undertook their investigations principally on rodents (albino mice, albino rats and New Zealand rabbits) and on some dogs but they also correlated the data available on other species. It was demonstrated that sulfur and nitrogen mustards given by different routes of administration absorbed readily, inducing injury to the lymphatic tissue, spleen, bone marrow and epithelium of the small intestine, and, if the dose was large enough, death of the animal. In general, the destructive action and the extensive alterations produced showed an extraordinarily close parallelism to the well known effects of the direct application of the roentgen

[1] Krumbhaar, E. B. Role of the blood and the bone marrow in certain forms of gas poisoning. *J. A. M. A.*, 1919, *72*, 39–41.

[2] Pappenheimer, A. M., and Vance, M. The effects of intravenous injections of dichloroethylsufide in rabbits with special reference to its leucotonic action. *J. Exper. Med.*, 1920, *31*, 71–94.

[3] Gilman, A. and Philips, F. S. The biological actions and therapeutic applications of the B-chloroethyl amines and sulfides. *Science*, 1946, *103*, 409–415; also, Philips, F. S., and Gilman. A. The relation between chemical constitution and biological action of the nitrogen mustards. Approaches to tumor chemotherapy. Am. Assoc. Advancement Sc., 1947, pp. 293–305.

[4] Graef, I. Karnofsky, D. A., Jager, V. B., Krichesky, B., and Smith, H. W. The clinical and pathologic effects of the nitrogen and sulfur mustards in laboratory animals. *Am. J. Path.*, 1948, *24*, 1–47.

rays. The authors stress, however, that for the moment, this parallelism must be regarded as only superficial since in neither case has the fundamental mode of injury been determined.

Gilman and Philips[3] in describing the pharmacology of the nitrogen mustards consider that the presence of the imine ring is important in endowing the compound with cytotoxic activity. However, the specific sites of action of the imine ring are dependent upon the rate of intramolecular cyclization of the parent substance and the formation of the imonium moiety. The end result is complete dissolution of the cell if the dose is large and varying degrees of growth inhibition if the dose is smaller.

Fell and Allsopp[5] studied the action of mustard gas ($\beta\beta'$ dichlorodiethylsulphide) on living cells in vitro by using as cultures small pieces of choroid and sclerotic from ten to twelve day chick embryos grown by the hanging drop method. It was noted that liquid mustard gas or its concentrated vapor immediately coagulated and killed living cells with little distortion of form. Low concentrations of mustard gas vapor led to slow death of the cells with disintegration of the cystoplasm associated with enormous distortion of the cell outline, of the chromosomes in mitotic cells and of the chromatin structure of resting nuclei. Cells grown in sublethal concentration of mustard gas showed many abnormal mitotic figures.

The histopathologic effect of the nitrogen mustard (HN_2) on tumors and other tissues in the human was analyzed by Spitz.[6] This author during a period of two and one-half years performed postmortem examinations in a group of 57 cases which were treated at Memorial Hospital principally with nitrogen mustard after other forms of therapy, including external irradiation, became no longer effective. The cases represented 12 of Hodgkin's disease, 16 of lymphosarcoma, 9 of lymphatic leukemia, 7 of myelogenous leukemia and 13 of a variety of malignant tumors.

The changes observed on the neoplastic tissues were, curiously, most pronounced in the lymphosarcomas although the most encouraging clinical results have been obtained in Hodgkin's disease. These changes consisted of ballooning of the cytoplasm by fat and of swelling with loss of chromatin pattern of the nuclei. They were attributed to the direct effect of the nitrogen mustard since no other known condition would lead to such accumulation of the lipid material. The destructive phase persisted for as long as twenty-one days following therapy in lymphosarcoma but in Hodgkin's disease it was not evident after an interval of eight days. In most cases, after this period, there was a transformation of the original tumor into one of steadily decreasing cell population with increased pleomorphism of the remaining cells, multinucleated giant cells making their appearance quite frequently. Not all cells were affected equally and some escaped injury altogether. In the lymphatic leukemias no cytologic alterations were observed; there was simply a reduction in the number of cells comprising the leukemic infiltrate. The myelogenous leukemias, except the acute cases, remained uninfluenced by the nitrogen mustard gas therapy. Likewise no cytologic effect was noted in a variety of malignant epithelial tumors.

Of the normal tissues, Spitz found changes attributable to the nitrogen mustard therapy in the bone marrow and the testes. The alterations of the bone marrow consisted of hypoplasia, disappearance of the granulocytes and persistence of a few erythroblasts and megakaryocytes. In the more severe cases hemorrhage, edema and diffuse myxomatous changes were also present. In general, the degree of hypoplasia of the bone marrow depended on the total cumulative dose and the interval following therapy. The effect noted in the testes was rather surprising. In no less than

[5] Fell, Honor B., and Allsopp. C. B. The action of mustard gas on living cells in vitro. *Cancer Research*, 1948, *8*, 145–161.

[6] Spitz, Sophie. The histological effects of nitrogen mustards on human tumors and tissues. *Cancer*, 1948, *1*, 383–398.

90 per cent of the cases examined a moderately advanced atrophy developed from which recovery seldom occurred. By comparison, testicular atrophy was found in only 38 per cent of miscellaneous malignant tumors and 57 per cent of malignant lymphomas and leukemias not subjected to mustard but to other forms of therapy. Spitz also made the important observation that in contradistinction to animal experiments the mucosa of the gastrointestinal tract of the human exhibited no unusual alterations, regardless of the amount or duration of the nitrogen mustard therapy.

The clinical value of nitrogen mustard therapy was studied by a number of investigators. Because of the above enumerated reasons, Hodgkin's disease, lymphosarcoma, the leukemias and a few other types of malignant neoplasms were chosen as most suitable for treatment. In the great majority of cases reported in the literature HN_2 was used. The average dose amounted to 0.1 mg. per kilogram of body weight which was repeated daily, for four to six doses. The drug was prepared fresh and to avoid local thrombosis it was injected within five minutes into the tube of a saline intravenous system already in operation. After intervals of two to three months further courses of treatment were given, depending on the symptoms of the patient.

Recently, Wintrobe and Huguley[7] dealing with a larger series of cases have critically evaluated what one may expect from such a procedure. These authors treated with HN_2 a total of 102 cases and have observed the response for periods of three to thirty-three months. They have also analyzed for the purpose of comparison the available cases from the literature. The best results were obtained in Hodgkin's disease. A regression of the tumefactions, splenomegaly, fever, bone pain and various other clinical symptoms was noted lasting repeatedly over periods of many months. In view of the fact that most of the cases treated were already radiation "resistant" or very far advanced, the marked palliative effect was considered as a notable accomplishment. In lymphosarcoma or reticulum cell sarcoma the results have not been so promising, but the authors cite several instances from the literature in which the outcome was more favorable. In chronic myelogenous leukemia and the asymptomatic stage of chronic lymphatic leukemia the effectiveness of HN_2 was often quite impressive. In the acute leukemias only very temporary alterations were obtained. The other malignant neoplasms treated likewise exhibited indefinite response. Ultimately, nitrogen mustard therapy proved ineffective in all lesions.

According to Wintrobe and Huguley, there are two disadvantages connected with the clinical application of the mustard therapy: (1) the nausea and vomiting which nearly always follows the first injection of each course, and (2) the depressant effect on the cellular elements of the blood resulting in lymphocytopenia, granulocytopenia, thrombocytopenia and some anemia. However, in the cases responding favorably this latter effect is short-lived and does not produce serious symptoms.

To eliminate some of these disadvantages Burchenal[8] tried the use of a new type of nitrogen mustard: the so-called Sk_{136} (1:3 propane diamine NNN'N' tetrakis (2-chloroethyl) dihydrochloride). Eleven patients with chronic myelogenous leukemia were treated with this drug. In general, Sk_{136}, when given in a dosage of 0.1 mg. per kilogram of body weight daily for four to eight doses, was found to cause much less nausea and vomiting than HN_2 but otherwise there was no difference in the clinical response. In 19 patients with acute leukemia SK_{136} remained largely unsatisfactory.

The question arose from the beginning of the clinical use of nitrogen mustard therapy whether or not its simultaneous applica-

[7] Wintrobe, M. M., and Huguley, C. M., Jr., Nitrogen mustard therapy for Hodgkin's disease, lymphosarcoma, the leukemias, and other disorders. *Cancer*, 1948, *1*, 357–382.

[8] Burchenal, J. H. The newer nitrogen mustards in the treatment of leukemia. *Radiology*, 1948, *50*, 494–499.

tion with roentgen therapy or other forms of irradiation may not be of advantage. Karnofsky, Burchenal, Ormsbee, Cornman and Rhoads[9] studied the additive toxicological effects of HN_2 and roentgen rays experimentally in mice. These experiments suggested that under certain conditions the destructive action of the two agents is at least additive. The impression was gained that the administration of a full course of HN_2 must precede the roentgen therapy to secure the best result. However, the authors emphasized that explorations of a much wider range are necessary before any definite recommendations can be made on this problem.

Since then several reports have appeared in the literature referring to various combinations of nitrogen mustard and roentgen therapy. In view of the clinical vagaries of the diseases for which the method is used and the very indefinite duration of the remissions that follow the treatment, however, it is still impossible to properly evaluate the cumulative effect of the two agents.

Their relative merit is much better understood. Craver,[10] on the basis of over 300 cases treated at Memorial Hospital and after reviewing the literature, summed this up as follows: (1) nitrogen mustard therapy is indispensable in (a) cases of generalized Hodgkin's disease with marked constitutional symptoms; (b) advanced cases of lymphosarcoma in which there is an immediate threat to life and in which the lesion causing the immediate danger is not amenable to surgery or irradiation; (c) anaplastic carcinoma of the lung; (2) in early and intermediate stages of Hodgkin's disease, in most cases of lymphosarcoma and in the majority of the chronic leukemias it seems doubtful that nitrogen mustard therapy offers any advantage or is as good an all around agent as roentgen therapy; and (3) none of the nitrogen mustards so far used has given any indication of ability to cure any of the types of cancer treated.

It may be said, as a general conclusion, that nitrogen mustard therapy has a marked palliative value. It undoubtedly will form an important adjunct of our future armamentarium in the treatment of certain well selected lesions. The trial of new compounds and the extension of the field of clinical exploration seems amply justified.

T. Leucutia, M.D.

Harper Hospital
Detroit 1, Mich.

[9] Karnofsky, D. A., Burchenal, J. H., Ormsbee, R. A., Cornman, I., and Rhoads, C. P. Experimental observations in the use of the nitrogen mustards in the treatment of neoplastic disease. Approaches to Tumor Chemotherapy. Am. Assoc. Advancement Sc., 1947, pp. 293–305.

[10] Craver, L. F. The nitrogen mustards: clinical use. *Radiology*, 1948, *50*, 486–493.

ELDWIN R. WITWER
1890-1948

IN THE death of Dr. Eldwin R. Witwer, which occurred from a cerebral hemorrhage on November 2, 1948, the medical profession lost one of its abler physicians and radiology lost one of its staunchest champions in the fight to prevent encroachment on its rights and privileges as a speciality. His position in regard to issues of ethics, high scientific standards and economic justice was never in doubt, and his decided views were influential on the floor of scientific meetings as well as in private counsel and committee sessions.

Dr. Witwer was born in Ontario, Canada,

on August 23, 1890, but was educated in Michigan schools, Valparaiso University and the Detroit College of Medicine, now Wayne University College of Medicine. His earlier interests were in pathology and internal medicine and he held the position of Assistant Professor of Pathology in his Alma Mater, where he later became Assistant Professor of Radiology. For many years he was medical assistant to Dr. C. G. Jennings, an internist of national repute, and his excellent training in pathology and internal medicine was of course the finest possible basis for diagnostic and therapeutic radiology.

After a year of special study in the Department of Radiology at Cook County Hospital, Chicago, Dr. Witwer became radiologist to Jennings Hospital in Detroit and in 1927 he became associated with Drs. William A. Evans and Lawrence Reynolds, joining the staff at Harper Hospital. Although his major professional career was as radiologist to Harper Hospital, he also served in similar capacity on the staffs of Cottage, Bon Secours, U. S. Marine and Children's Hospitals.

His militant crusading spirit kept him in the vanguard of medical organizational activities. He was prominent in the conduct of the affairs of the local and state medical societies and had much to do with the policies of establishing and conducting the Michigan Medical and Hospital Services under the Blue Cross plan, serving as trustee of that organization. Because of his talent and willingness to work he was the perennial secretary of the Detroit Roentgen Ray and Radium Society, holding this position for twenty-two years, interrupted only by the time which he served as its president. The Radiological Society of North America honored him with its presidency for the year of 1945–1946. The American Roentgen Ray Society, the American College of Radiology, of which he was chancellor, and the American Medical Association all made use of his knowledge and industry in appointment to important committees. He was admitted to membership in the American Roentgen Ray Society in 1928 and was among the group certified as radiologists by the American Board of Radiology in 1934. The Rocky Mountain Radiological Society and the Indiana State Medical Society elected him to honorary membership.

Much could be written about Dr. Witwer's extra-medical activities, for they were many. An enthusiastic farmer, he belonged to a number of farm clubs and he was owner and breeder of fine blooded stock. An active Mason, he belonged to several masonic orders and was president of the Detroit Masonic Temple Association. His friends and acquaintances were legion and included farmers, tradesmen and laborers as well as captains of industry, and leaders in the professional world. He is survived by his wife, Maude Gibbing Witwer, and a daughter, Mary Ellen Witwer.

E. Walter Hall, M.D.

JOSÉ FRANCISCO MERLO GÓMEZ

WORD has come of the death on September 28, 1948 of Dr. José Francisco Merlo Gómez, one of the leading radiologists in South America. Dr. José F. Merlo Gómez was for many years an active member of the military establishment of the Argentine and Professor of Radiology in the Military Medical School. He graduated from the Faculty of the Medical School in Buenos Aires in 1912. He took post-graduate work in Europe, part of the time in Paris as an externe during the first world war. He served as assistant, then as head of the service in radiology in the Hospital de la Pitié.

In Argentina he served as chief of the x-ray service of the Surgical Clinic in the Hospital Nacional de Clinicas, as well as in a private sanitarium.

Dr. Merlo Gómez was President of the First Inter-American Congress of Radiology in Buenos Aires in 1943, and Honorary President of the Second Inter-American Congress of Radiology held in Havana in 1946.

Dr. Merlo Gómez was also a corresponding member of the Academy of Medicine of Colombia; honorary member of the American College of Radiology; Honorary President of the Inter-American College of Radiology, which he was chiefly instrumental in organizing; honorary member of the Cuban, Mexican, Colombian, and Peruvian radiological societies, and President of the Argentinian Society of Radiology.

Many of us here in the United States will recall with pleasure the visit of Dr. Merlo Gómez and his delightful family following the Havana meeting in 1946, and share the sorrow of Señora Gómez and their children.

James T. Case, M.D.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: Netherland Plaza Hotel, Cincinnati, Ohio, Oct. 4–7, 1949.

American Radium Society
Secretary, Dr. H. F. Hare, 605 Commonwealth Ave., Boston, Mass. Annual meeting: Ambassador Hotel, Atlantic City, N. J., June 5–7, 1949.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: 1949, to be announced.

American College of Radiology
Executive Secretary, William C. Stronach, 20 N. Wacker Drive, Chicago 6. Annual meeting: Chalfonte-Haddon Hall, Atlantic City, N. J., June 5, 1949.

Section on Radiology, American Medical Association
Secretary, Dr. U. V. Portmann, Cleveland Clinic, Cleveland, Ohio, Annual Meeting: Atlantic City, N. J., June 8–10, 1949.

Alabama Radiological Society
Secretary, Dr. W. W. Anderson, Tuscaloosa, Ala. Next meeting time and place of Alabama State Medical Association,

Arkansas Radiological Society
Secretary, Dr. Fred Hames, 511 National Bldg., Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Brooklyn Roentgen Ray Society
Secretary, Dr. A. H. Levy, 1354 Carroll St., Brooklyn 13, N. Y. Meets monthly on fourth Tuesday, October to April.

Buffalo Radiological Society
Secretary, Dr. Mario C. Gian, 610 Niagara St., Buffalo, N. Y. Meets second Monday evening each month, October to May inclusive.

Central New York Roentgen Ray Society
Secretary, Dr. Dwight V. Needham, 608 E. Genesee St., Syracuse N. Y. Meets January, May, November.

Central Ohio Radiological Society
Secretary, Dr. Paul D. Meyer, Grant Hospital, Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, December, February, April, and June at Seneca Hotel, Columbus, Ohio.

Chicago Roentgen Society
Secretary, Dr. John H. Gilmore, 720 N. Michigan Ave., Chicago 11, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. Eugene L. Saenger, 735 Doctors Bldg., Cincinnati 2, Ohio. Meets last Monday of each month, September to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. George L. Sackett, 10515 Carnegie Ave. Cleveland 6, Ohio. Meetings at 6:30 P.M. on fourth Monday of each month from October to April.

Colorado Radiological Society
Secretary, Dr. Mark S. Donovan, 306 Majestic Bldg., Denver 2, Colo. Meets third Friday of each month at Department of Radiology, Colorado School of Medicine.

Connecticut Valley Radiologic Society
Secretary, Dr. E. W. Godfrey, 1676 Boulevard, West Hartford, Conn. Meets second Friday of October and April.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meets in Dallas on odd months and in Fort Worth on even months, and third Monday, 7:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. W. G. Belanger, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434-30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

Florida Radiological Society
Secretary, Dr. F. K. Hurt, Riverside Hospital, Jacksonville, Fla. Meets twice annually, in the spring with the annual State Society meeting, and in the fall.

Georgia Radiological Society
Secretary, Dr. Robert Drane, DeRenne Apartments, Savannah, Ga. Meets in mid-winter and at annual meeting of Medical Association of Georgia in the spring.

Houston X-ray Club
Secretary, Dr. Curtis H. Burge, 3020 San Jacinto St., Houston 4, Texas. Meets fourth Monday each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. William M. Loehr, 712 Hume-Mansur Bldg., Indianapolis 4. Meets second Sunday in May.

Iowa X-Ray Club
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. Special meetings by announcement.

Kansas Radiological Society
Secretary, Dr. Anthony F. Rossitto, Wichita Hospital, Wichita, Kan. Meets annually with State Medical Society.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

Long Island Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. Moris Horwitz, 441 No. Camden Drive, Beverly Hills, Calif. Meets second Wednesday each month at Los Angeles County Medical Assn. Building.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

Louisville Radiological Society
Secretary, Dr. E. L. Pirkey, Louisville General Hospital, Louisville 2, Ky. Meets monthly on second Friday at Louisville General Hospital.

* Secretaries of societies not here listed are requested to send the necessary information to the Editor.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. R. D. McDuff, 220 Genesee Bank Bldg., Flint 3, Mich.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Chauncey N. Borman, 802 Medical Arts Bldg., Minneapolis 2, Minn. Two meetings yearly, one at time of Minnesota State Medical Association the other in the fall.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. Ralph C. Moore, Nebraska Methodist Hospital, Omaha 3, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. George Levene, Massachusetts Memorial Hospitals, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

NORTH CAROLINA RADIOLOGICAL SOCIETY
Secretary, Dr. J. E. Hemphill, 323 Professional Bldg., Charlotte 2, N. C. Meets in May and October.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. C. O. Heilman, 807 Broadway, Fargo. Meetings held by announcement.

NORTHERN CALIFORNIA RADIOLOGICAL CLUB
Secretary, Dr. C. E. Grayson, Medico-Dental Bldg., Sacramento 14, Calif. Meets at dinner last Monday, every second month, except June, July and August. Next meeting Sept. 27, 1948.

OHIO STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Carroll C. Dundon, 2065 Adelbert Road, Cleveland 6, Ohio.

OKLAHOMA STATE RADIOLOGICAL SOCIETY
Secretary, Dr. W. E. Brown, Tulsa, Okla. Three regular meetings annually.

OREGON RADIOLOGICAL SOCIETY
Secretary, Dr. Boyd Isenhart, 214 Medical Dental Bldg., Portland 5, Oregon. Meets monthly 2nd Wednesday, 8:00 P.M., Library of University of Oregon Medical School.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY
Secretary, Dr. S. J. Hawley, 1320 Madison St., Seattle 4, Wash. Meets annually in May.

PACIFIC ROENTGEN SOCIETY
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. J. M. Converse, 416 Pine St., Williamsport.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. Arthur Finkelstein, Graduate Hospital, 19th and Lombard St. Meets first Thursday each month October to May, at 8:00 P.M., in Thomson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. R. P. Meader, 4002 Jenkins Arcade Pittsburgh 22, Pa. Meets 6:30 P.M. at Webster Hall Hotel on second Wednesday each month, October to May inclusive.

QUEENS ROENTGEN RAY SOCIETY
Secretary, Dr. J. E. Goldstein, 88-29 163rd St., Jamaica 3, N. Y. Meets fourth Monday of each month except during the summer.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Harry A. Miller, 2452 Eutaw Place, Baltimore. Meets third Tuesday each month, September to May.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Robert M. Lowman, Grace-New Haven Community Hospital, New Haven 11. Meets bimonthly second Thursday, at place selected by Secretary.

RADIOLOGICAL SECTION, DISTRICT OF COLUMBIA MEDICAL SOCIETY
Secretary, Dr. A. A. J. Den, 1801 K St., N. W., Washington, D. C. Meets Medical Society Auditorium, third Thursday, January, March, May, October at 8:00 P.M.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. Raphael Pomeranz, 31 Lincoln Park, Newark, N. J. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. Murray P. George, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary, Dr. Maurice D. Frazer, 1037 Stuart Bldg., Lincoln, Nebr.

ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary, Dr. Edwin C. Ernst, Beaumont Medical Building, St. Louis, Mo. Meets fourth Wednesday of each month, except June, July, August, and September.

SAN DIEGO ROENTGEN SOCIETY
Secretary, Dr. R. F. Niehaus, 1831 Fourth Ave., San Diego, Calif. Meets monthly, first Wednesday at dinner.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. D. R. MacColl, 2007 Wilshire Blvd., Los Angeles 5, Calif.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. Harold L. Shinall, St. Joseph's Hospital, Bloomington, Ill.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

SOUTH CAROLINA X-RAY SOCIETY
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

TENNESSEE RADIOLOGICAL SOCIETY
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

TEXAS RADIOLOGICAL SOCIETY
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth 4, Texas. Next meeting Jan. 7–8, 1949, Fort Worth, Texas.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May inclusive. Room 203, Service Memorial Institute, 426 N. Charter St., Madison.

Utah State Radiological Society
Secretary, Dr. Angus K. Wilson, 343 S. Main St., Salt Lake City 1, Utah. Meets third Wednesday in September, November, January, March and May.

Virginia Radiological Society
Secretary, Dr. P. B. Parsons, Norfolk General Hospital, Norfolk, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Homer V. Hartzell, 310 Stimson Bldg., Seattle 1, Wash. Meets fourth Monday each month, October through May, College Club, Seattle.

X-Ray Study Club of San Francisco
Secretary, Dr. Ivan J. Miller, 49 Fourth St., San Francisco 3. Meets monthly on third Thursday at 7:45 P.M., January to June at Lane Hall, Stanford University Hospital, and July to December at Langley Porter Clinic, University of California Hospital.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

Mexico

Sociedad Mexicana de Radiologia y Fisioterapia
General Secretary, Dr. D. P. Cossio, Marsella No. 11, Mexico, D. F. Meets first Monday of each month.

British Empire

British Institute of Radiology Incorporated with the Roentgen Society
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

Faculty of Radiologists
Honorary Secretary, Dr. J. F. Bromley, 45, Lincoln's Inn Fields, London, W.C.2, England.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London.

Canadian Association of Radiologists
Honorary Secretary, Dr. E. M. Crawford, 2100 Marlowe Ave., Montreal 28, Que. Meetings January and June.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Francaise d'Electrologie et de Radiologie Médicales
Secretary, Dr. Origéne Dufresne, 4120 Ontario St., East, Montreal, P. Q.

Australian and New Zealand Association of Radiologists
Honorary Secretary, Dr. Alan R. Colwell, 135 Macquarie St., Sydney, N.S.W.
Honorary Secretaries, State Branches:
New South Wales, Dr. E. W. Frecker, 135 Macquarie St., Sydney.
Victoria, Dr. T. L. Tyrer, 3 Lockerbie Court, East St. Kilda.
Queensland, Dr. J. Adam, 131 Wickham Terrace, Brisbane.
South Australia, Dr. B. C. Smeaton, 178 North Terrace, Adelaide.
Western Australia, Dr. A. M. Nelson, 179-B St. Georges Terrace, Perth.
New Zealand, Dr. E. G. Lynch, 12 Bolton St., Wellington.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Sociedade Brasileira de Radiologia Medica
Secretary, Dr. Nicola Caminha, Av. Mem de Sa, Rio de Janeiro, Brazil. Meets monthly, except during January, February and March.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Andrelino Amaral, Av. Brigadeiro Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Tuesday at 9 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Peruana de Radiologia
Secretary, Dr. Julio Bedoya Paredes, Apartado, 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta, 218, Lima.

Sociedad de Radiologica, Cancerologia y Fisica Medica del Uruguay
Secretary, Dr. Arias Bellini.

Continental Europe

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 111 Avenue des Alliés, Louvain, Belgium. Meets monthly, second Sunday at Maison des Médecins, Brussels.

Ceskoslovenská společnost pro röntgenologii a radiologii v Praze
Secretary, Dr. Roman Bláha, Praha XII, stát. nemocnice, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland. Next meeting, Krakow, June 2 and 3, 1949.

Gdansk Section, Polish Society of Radiology
Secretary, Dr. A. Smigielska, Akademia Lekarska, Gdansk. Meets monthly last Sunday at 10.30, X-Ray Dept., Akademia Gdansk.

Warsaw Section, Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland. Meets monthly.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad. USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Schweizerische Röntgen-Gesellschaft (Société Suisse de Radiologie)
President, Dr. H. E. Walther, Gloriastr. 14, Zürich, Switzerland.

Societa Italiana di Radiologia Medica
Secretary, Prof. Mario Ponzio, Ospedale Mauriziano Torino, Italy. Meets biannually.

THE EASTERN CONFERENCE OF RADIOLOGISTS

The Eastern Conference of Radiologists is to be revived in the Spring of 1949. The Conference, previously known as the Mid-winter Conference of Eastern Radiologists, was suspended in 1940 due to World War II. Five cities participate, namely, Boston, New York, Philadelphia, Baltimore and Washington and it was the unanimous recommendation of the radiological societies in the respective cities that the Eastern Conference be revived. Washington, D. C., is to be the host city for a meeting on Friday and Saturday, March 11 and 12, 1949, with headquarters at the Hotel Statler. All radiologists are invited.

On the *pink insert* in the advertising section will be found detailed information concerning the meeting, with a registration form on the reverse side of the page. Those in charge of arrangements wish to gain an idea of the possible attendance at the meeting and they will appreciate having this registration form made out and sent in as soon as possible by those who plan to attend the meeting.

Below is given the detailed scientific program which has been arranged for the meeting of the Eastern Conference of Radiologists.

Friday Morning, March 11, 1949

Presidential Ballroom, Hotel Statler

9:00–10:15 A.M. PANEL DISCUSSION

The Correlation of Electro-Encephalography, Pneumo-Encephalography and Arteriography in the Diagnosis of Lesions of the Brain.

1. Roentgenological Aspects—George M. Wyatt, M.D., Moderator
2. Neurosurgical Aspects—Arthur A. Morris, M.D., Neurosurgeon, Doctors Hospital.
3. Neurological Aspects—Robert H. Groh, M.D., Associate in Neurology, Department of Neurology and Psychiatry, George Washington University Medical School.
4. Neuropathological Aspects—Webb Haymaker, M.D., Chief of Neurological Pathology Section, Army Institute of Pathology.

10:15–10:30 A.M. Intermission

10:30–10:45 A.M.

Supervoltage Radiation Therapy of Brain Tumors. Major Keene M. Wallace, M.C., Chief of Radiation Therapy, Walter Reed General Hospital.

10:45–11:00 A.M.

Congenital Vascular Anomalies (Phlebectasia). Captain Acors W. Thompson, M.C., Chief of Peripheral Vascular Section, Walter Reed General Hospital.

11:00–11:15 A.M.

Diagnostic Problems in the Normal and Pathological Physiology of the Colon. Dr. Willy E. Baensch, Professor of Roentgenology, Georgetown University Medical School and Hospital.

11:15–11:30 A.M.

Radiologist's Role in the Prevention and Early Diagnosis of Cancer of the Colon. Fred O. Coe, M.D., Radiologist. From the Radiological Clinic of Drs. Groover, Christie and Merritt.

11:30–11:45 A.M.

Differential Diagnosis of Inflammatory, Benign and Malignant Extrinsic Lesions of the Gastrointestinal Tract. Henry W. Wood, M.D., Fellow in Radiology. From the Radiological Clinic of Drs. Groover, Christie and Merritt.

Discussed by: Aubrey O. Hampton, M.D., Chief Radiologist, Garfield Memorial Hospital.

11:45–12:00 A.M.

Colon and Small Bowel Injury Following 1000 kv Roentgen Therapy. Colonel Harold I. Amory, M.C., Chief Radiologist, and Irving D. Brick, M.D., Consultant in Gastroenterology, Walter Reed General Hospital.

12:00–12:15 P.M.

A New Technique for Radium Therapy in Carcinoma of the Bladder. Milton Friedman, M.D., Consultant in Radiation Therapy, Walter Reed General Hospital, and Lloyd G. Lewis, M.D., Professor of Urology, Georgetown University Medical School.

12:15–12:30 P.M.

Primary Ureteral Tumor; a Case Report. William M. Clopton, M.D., Radiologist, Sibley Memorial Hospital.

12:30–2:30 P.M. Luncheon Period.

Friday Afternoon, March 11, 1949

National Bureau of Standards and The National Cancer Institute, United States Public Health Service

This part of the program will be held at the National Bureau of Standards. Buses from the Statler Hotel to the Bureau will be provided.

2:30–4:30 P.M.

Biological Effects of Chronic Irradiation with Gamma Rays on Laboratory Animals. By the Staff of the National Cancer Institute.

1. Introductory Remarks (5 min.). Egon Lorenz, Ph.D.
2. Effects on Breeding Behavior of Mice (20 min.). Margaret K. Derringer, Ph.D.
3. Pathology of Radiation Injury (20 min.). Allen B. Eschenbrenner, Ph.D.
4. Effects on Life Span, Hematopoiesis and Carcinogenesis (20 min.). Egon Lorenz, Ph.D.

Physical Aspects of X-ray Protection and Dosage Measurement. By the Staff of the National Bureau of Standards.

5. Conditions for the Economical Achievement of X-radiation Protection (20 min.). Harold O. Wyckoff, Ph.D.
6. Calibration Use of Thimble Chambers up to 1,400 kv. (20 min.). Lauriston S. Taylor, Ph.D.
7. Conditions for the Proper Utilization of Dosage Meters, Particularly in the Low Energy Range (20 min.). Frank H. Day, M.S.
8. Inspection of the Bureau of Standards' two new high voltage radiation laboratories.

Following the presentation of the papers the X-ray Section will hold an "open house" in its recently completed 1,400 kilovolt x-ray laboratory. In this will be shown the 1,400 kv. x-ray generator, x-ray tube, ion tube and electron tube, together with the special high pressure ionization chambers used with them. There will also be included the standardization laboratory for calibrating thimble chambers up to 200 kilovolts and for low voltage high intensity radiations such as obtained from contact therapy and beryllium-window tubes. The new multimillion-volt betatron laboratory will also be open for inspection. There are numerous other laboratories about the Bureau working on problems of direct or indirect interest to the radiologist.

6:30–8:00 P.M. Cocktails, Congressional Ballroom

8:00 P.M. Banquet, Presidential Ballroom

Saturday, March 12, 1948

Congressional Ballroom, Hotel Statler

9:00–10:15 A.M. PANEL DISCUSSION

The Management of "Surgically Incurable" Cancer of the Breast; a discussion devoted to the following specific aspects of the disease:

1. Local Lesions
2. Regional Nodes
3. Visceral Metastasis
4. Osseous Metastasis

Ralph M. Caulk, M.D., Moderator, Chief of Radiation Therapy, Garfield Memorial Hospital.

1. Surgical Aspects—Calvin T. Klopp, M.D., Medical Director, Warwick Memorial Clinic.
2. Radiation Therapy—U. V. Wilcox, M.D., Radiation Therapist, Doctors Hospital.
3. Endocrine Aspects—Roy Hertz, M.D., Chairman Endocrinology Section, National Cancer Institute; Associate, Warwick Memorial Clinic.
4. Neurosurgical Aspects—James W. Watts, M.D., Associate Professor of Neurosurgery, George Washington University Medical School.

10:30–10:45 A.M.

Tissue Distribution of some Organic Compounds of Radioisotopes. Charles F. Geschickter, M.D., Professor of Pathology, Georgetown University Medical School and Hospital.

10:45–11:00 A.M.

Prenatal Bowing of Tubular Bones; a Presentation of Cases. Francis C. Allman, M.D., Associate Radiologist, Providence Hospital.

11:00–11:15 A.M.

Orbital Foreign Bodies; Criteria for Detection and Discussion of the Pfeiffer Method for Precise Localization. Alfred A. J. Den, M.D., Radiologist, Episcopal Eye, Ear and Throat Hospital.

11:15–11:30 A.M.

Epilation in Ringworm of the Scalp (Microsporum auidini). Isidore Lattman, M.D., Radiologist, Children's Hospital.

11:30–11:45 A.M.

Intussusception; Reduced by Barium Enema (Hydrostatic Pressure). Isidore Lattman, M.D., Radiologist, and Gerald H. McAteer, M.D., Attending Surgeon, Children's Hospital.

11:45–12:00 A.M.

An Analysis of Approximately 7,000 "Positive" Chest Roentgenograms Encountered in a Recent Survey in the District of Columbia. Rollin F. Bunch, M.D., Fellow in Radiology. From the Radiological Clinic of Drs. Groover, Christie and Merritt.

Discussed by: Aubrey O. Hampton, M.D., Chief Radiologist, Garfield Memorial Hospital.

12:00–12:15 P.M.

Value of Chest Roentgenograms as a Routine Part of Cancer Detection Examinations. C. H. Wallman, M.D., Fellow in Radiology. From the Radiological Clinic of Drs. Groover, Christie and Merritt.

Discussed by: Aubrey O. Hampton, M.D., Chief Radiologist, Garfield Memorial Hospital.

12:15–12:30 P.M.

The Clinical Application of Radioisotopes in the Diagnosis and Treatment of Cancer. Murray M. Copeland, M.D., Professor of Oncology and Radiotherapist, Georgetown University Hospital.

12:30–12:45 P.M.

Results of Radiation Therapy alone in Adenocarcinoma of the Uterine Cervix and Corpus. George D. Adams, M.D., Fellow in Radiology. From the Radiological Clinic of Drs. Groover, Christie, and Merritt.

THIRD INTER-AMERICAN CONGRESS OF RADIOLOGY

The Third Inter-American Congress of Radiology will be held in Santiago, Chile, from November 11 to November 17, 1949. Prior announcements of the meeting (see especially this Journal, page 559, October, 1948) have elicited many inquiries requesting information regarding transportation to and from Santiago.

The radiologist who has ample time and would enjoy an ocean voyage can sail from New York or New Orleans and arrive in Rio de Janeiro twelve days later. A program of visits to medical institutions and scenic high spots can be arranged in Rio; thence to Sao Paulo, via Pan American World Airways, visiting Montevideo, Buenos Aires, the Chilean lakes district, and Santiago.

After the Congress has ended, a northern flight can be arranged with stops at Arequipa, the Incaland Country, Lima, Quito, Cali, Bogota, Medellin, Balboa, C. Z., Guatemala, Merida, Yucatan, Mexico, and finally New Orleans.

Physicians who are pressed for time but who would like to attend the Santiago meeting may fly directly from New York, New Orleans, Miami, or Mexico City, to Santiago.

Special itineraries will be made for those who wish to attend the Congress, and will be personalized to include such stops as are chosen.

Dr. James T. Case, 55 East Washington Street, Chicago 2, Illinois, chairman of the Committee on International Affairs of the American College of Radiology, will be happy to assist all interested radiologists in making their plans. Because of the great demand for South American transportation during the winter months, all those who foresee a possibility of attending the Third Inter-American Congress of Radiology should write Doctor Case immediately.

THE AMERICAN BOARD OF RADIOLOGY

Certification of Radiation Physicists

Beginning January 1, 1949, The American Board of Radiology will examine and certify physicists as radiation physicists. Three types of certificates will be granted:

(1) Radiological Physics
(2) X-ray and Radium Physics
(3) Medical Nuclear Physics

(2) and (3) are both included in (1).

Each applicant for any one of the certificates in radiation physics will be required to meet the following standards:

(a) Satisfactory moral and ethical standing.

(b) That he holds himself to be a specialist in the category of physics designated in his application.

(c) That he be a citizen of the United States or Canada. Candidates from other countries must be permanent residents of that country and native citizens thereof.

(d) That he holds a degree of Bachelor of Arts or its equivalent and has majored in physical science or engineering.

(e) That he be a member of the American Physical Society or similar organization.

(f) That after graduation from college he has had at least one year postgraduate experience in radiation physics or in a radiation physics laboratory.

(g) Candidates for examination in Radiological Physics in addition to requirements specified in paragraphs (a), (b), (c), (d), (e) and (f) must have had at least one year of experience in association with a Department of Radiology approved by the Board and throughout one of the two years specified must have had experience in medical application of artificial radioactive materials.

Each candidate shall submit with his application personal records of calibrations of one low voltage and one high voltage x-ray apparatus, personal records of one x-ray protection survey and one protection survey for radioactive materials.

(h) Candidates for examination in X-ray and Radium Physics in addition to requirements specified in paragraphs (a), (b), (c), (d), (e) and (f) must have had at least one year of experience in association with a Department of Radiology approved by the Board. With the application he must submit records of personal calibration of one low voltage and one high voltage x-ray apparatus and personal records of one x-ray protection survey and one personal protection survey for radium.

(i) Candidates for examination in Medical Nuclear Physics in addition to requirements specified in paragraphs (a), (b), (c), (d), (e) and (f) must have had one year of experience in physical procedures relative to medical application of radioactive materials.

(j) Applications shall be endorsed by a Diplomate of the American Board of Radiology and a Diplomate in Radiological Physics who have personal knowledge of the experience, training, moral and ethical standing of the applicant and that he is qualified to take an examination.

(k) A fee of $25.00 shall accompany the application which will be refunded if the application for examination is not accepted.

(l) Applications shall be submitted to the Secretary of The American Board of Radiology. He shall forward them to the Credentials Committee on Radiological Physics to be appointed by the Board, who shall decide about the candidate's fitness to be examined. Those approved for examination shall be informed by the Secretary of the Board when and where to appear.

Examinations will usually be conducted at the time and place of the regular or special meetings of the American Board of Radiology.

Examinations in radiation physics shall be oral though practical or written examinations may be required. Examinations will be designed to test the candidate's knowledge and his fitness to practice physics in the category for which he applies. Accordingly, the examination may include questions regarding the structure of matter, construction and operation of x-ray apparatus, the use of radiation measuring instruments, determination of radiation quantity and quality, protection from x-rays, radium and artificial radioactive substances, preparation of radioactive applicators, determination of dosages of x-rays, radium and radioactive substances and other questions relative to the category in which the candidate applies.

Candidates who fail an examination shall not be admitted to another examination until one year has elapsed. They must request re-examination at least sixty days prior to the next meeting of the Board and pay an additional fee of $10.00.

A diploma may be revoked if in the opinion of the Board a misstatement of fact has been made in the application or any other communication to the Board or its representatives, or for expulsion from a scientific society for misconduct. Under such circumstances the diplomas must be returned to the Board and the name of the individual shall be omitted from the Registry of Diplomates.

Address all communications to the Secretary of the American Board of Radiology.

B. R. Kirklin, M.D., *Secretary*
American Board of Radiology
Mayo Clinic
Rochester, Minnesota

ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

Department Editor: GEORGE M. WYATT, M.D., 1835 Eye St., N.W., Washington 6, D. C.

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ROENTGEN DIAGNOSIS

NECK AND CHEST

Dock, William. Reasons for the common anatomic location of pulmonary tuberculosis. *Radiology*, April, 1947, *48*, 319–322.

As the cardiac output is less when the subject is sitting or standing than in recumbency, and pulmonary arterial pressure varies with the output, even lower pressure must occur in the right ventricles of persons who sit or move but little while on their feet. This means that the column of blood from the right heart to the apical part of the lungs exerts an equal or greater pressure. In view of the fall in pressure along the course of the vessel, there can scarcely be any blood flow, and no formation of lymph or tissue fluid at the upper third of the lungs under these conditions. Because of the length and tortuosity of the right pulmonary artery pressure on that side will be lower and the bloodless region larger than on the left.

As is well known primary lung lesions initiated prior to development of resistance are disseminated generally throughout the lungs of men, cattle, and rabbits. It seems probable that in animals or men with reinfection or with rapid development of high resistance an inadequate pulmonary blood flow, for many hours each day, may permit tuberculosis to progress in the lung field highest above the heart, while lower parts enjoy a considerable degree of immunity. In adults, progressive lesions observed soon after reversal of a negative skin test are usually in the upper lung field, and in the usual instances of "reinfection," apical disease progresses and the opposite apex becomes involved even though other regions, in spite of a constantly positive sputum, remain free for months, years, or decades.

One reason for accepting the probability that relative ischemia is the basis for the apical location of phthisis in man is the very low incidence of progressive apical lesions in patients with mitral stenosis and the very high incidence in those with pulmonic stenosis.

The significance of this theory, in treatment by bed rest and collapse is that absolute recumbency, not rest in the propped up or sitting posture, is the factor most important in management.—*James J. McCort, M. D.*

Doig, A. T., and McLaughlin, A. I. G. Clearing of x-ray shadows in welders' siderosis. *Lancet*, May 22, 1948, *1*, 789–791.

This is a follow-up study of the authors' original (1936) description of a siderosis due to inhalation of nearly pure iron oxide particles

in welding fumes. At that time they concluded that the opacity noted in roentgenograms might be due to the iron oxide alone, without the association of fibrosis and congestion. The confirmatory literature since that time is quoted, including the only case of a postmortem examination in a previously known welders' siderosis. No fibrosis was found in the lung tissue.

The present follow-up includes one man who quit welding because of his abnormal chest film. Follow-up films were unchanged for two years. The patient was lost track of until nine years after he was first seen. At this time there was complete clearing of the previously described siderosis.

A second man became an instructor of welding. At this occupation only one-fourth of his time was spent at active welding. His chest film showed considerable resolution of the original process.

These 2 cases provide evidence that the roentgenographic reticulation and nodulation of welders' siderosis is not necessarily permanent, and that iron oxide dust can be eliminated from the lung parenchyma after some years.—*J. S. Summers, M. D.*

WEARING, J. D. H. Bronchiectasis simulating chronic bronchitis; a study of 46 cases. *Lancet*, May 29, 1948, *1*, 822–824.

There is a large group of patients with bronchiectasis in whom the symptoms and physical signs closely resemble chronic bronchitis. During a period of eighteen months at a military hospital the author examined 211 men and 3 women with signs of a chronic bronchitis, but without signs and symptoms classically attributed to bronchiectasis. Bronchograms of each patient revealed 46 (21 per cent) to have bronchiectasis. There were 31 cases of cylindrical, 3 of varicose and 12 of saccular type bronchiectasis.

In 26 (56 per cent) of the patients with bronchiectasis there was a history of "pneumonia." In the 168 cases without bronchiectasis only 32 per cent gave a history of "pneumonia." The author believes that the bronchi in unresolved pneumonia at first undergo reversible mechanical dilatation which may later be rendered irreversible by secondary infection of the bronchial wall. A bronchus may remain dilated for several months and yet be capable of returning to its original diameter.

In certain forms of bronchiectasis, a plain film is difficult to interpret correctly. Only by bronchography is accurate diagnosis of bronchiectasis possible in the living subject.—*J. S. Summers, M.D.*

NORRIS, CHAS. M. Early clinical features of bronchogenic carcinoma; illustrative cases. *Dis. of Chest*, March-April, 1948, *14*, 198–217.

Analysis of 310 proved cases of bronchogenic carcinoma seen at Chevalier Jackson Bronchoscopic Clinic shows a histological diagnosis was made during life in 97 per cent, of which bronchoscopic biopsy yielded 71 per cent and aspiration biopsy 14.5 per cent. Early manifestations of bronchial carcinoma depend on the point of origin of the lesion. About 50 per cent arise in the main stem, or lobar bronchus; about 25 per cent each rise in the segmental bronchi, and peripheral branches. The author describes the clinical symptoms and physical findings correlating them with case reports and films of the origin of the tumor. The main bronchus lesion may give no positive roentgen findings, but in this series, only 6 of 310 chest films were thought to be essentially negative. The author stresses the value of bronchoscopy when symptoms persist and films are negative. The secondary effects of tumor are described such as bronchial obstruction with atelectasis or ball-valve emphysema, which is seen on fluoroscopy. Segmental bronchial tumors usually show a wedge-shaped atelectasis. The peripheral tumors are frequently "silent" and may not be discovered until considerable size gives rise to pulmonary symptoms. Aspiration biopsy in 69 cases gave a positive diagnosis of malignancy in 45, or 66 per cent.—*Fred R. Gilmore, M.D.*

HERBUT, PETER A., and CLERF, LOUIS H. Cancer cells in bronchial secretions. *Tuberculology*, Dec., 1947, *9*, 90–92.

The authors outline the method of examination of bronchial secretions obtained by the bronchoscope in an attempt to make an earlier diagnosis of lung carcinoma. Using the Papanicolaou technique they examined 525 preparations of which 118 cases are proved carcinoma; cancerous cells were present in 105 cases, or 89 per cent, while a positive bronchoscopic biopsy was made in 52 cases, or 44 per cent. However, in 32 cases, or 27 per cent, the bronchoscopic examination was completely negative, but neoplastic cells were found in the bronchial secretions. There are three types of

tumor cells classified: squamous, completely undifferentiated, and all others. The first group comprises cells in clumps or alone. These contain moderate cytoplasm with deeply staining pleomorphic nuclei; occasionally, there are typical pearls present. The undifferentiated group usually shows single cells with scanty cytoplasm and a round, deeply staining homogeneous nucleus. The last group varies widely. Nevertheless, when there are inflammatory or Langhans cells present, these do not imitate the neoplastic cells.—*Fred R. Gilmore, M.D.*

BLAIR, L. G. Interpretation of children's chest x rays. *Brit. J. Radiol.*, June, 1947, *20*, 223–237.

The first step in interpretation of the chest film of an infant is an assessment of the technical factors. If the child was rotated or if the exposure was made in some other phase than full inspiration, the heart and mediastinum are much broader and the lung markings are crowded, suggesting collapse; or there is generalized diminished translucency. If the exposure is made during rapid inspiration, part of the lung may actually appear consolidated. Films made in strenuous expiration with crying may show a mediastinum which is very wide and apparently grossly pathological. Exposures made supine with slight rotation show considerable displacement of the mediastinum, which, being very mobile, usually falls off the spine.

Although the pathological changes which occur in children's chests may be the same as those in adults, there is a large group of specifically infants' diseases which are to be kept in mind. Elevation of the whole of one diaphragm leaf might indicate increased intra-abdominal pressure, Petit's eventration, or subdiaphragmatic abscess. A portion of the diaphragm may be elevated because of localized muscle weakness with bulging in one area; liver cysts or abscesses produce a similar situation. Diaphragmatic hernia, congenitally short esophagus, or even absence of one leaf sometimes enter into the differential diagnosis and are to be considered.

The bony structures of the thorax reflect generalized bone diseases, and rickets and scurvy may be diagnosed in this manner. Xanthomatosis and bone tuberculosis are to be kept in mind in dealing with children. Bone destruction in the region of a tumor suggests an inflammatory process as the ribs in children give more readily than adult structures to pressure and a malignant tumor is more likely to displace the ribs than destroy them.

Fluoroscopy is important in demonstrating unusual shift of the mediastinum in bronchial obstruction. The mediastinum shifts toward collapse, consolidation, long-standing effusion, and also toward the side in which there is agenesis of a lobe or the lung. In the last instance there is emphysema on the side of the chest toward which the mediastinum shifts. Shift is away from a recent effusion and obstructive emphysema.

Enlargement of the mediastinum due to tuberculosis is indistinguishable from that due to Hodgkin's disease or lymphosarcoma. Other mediastinal enlargements are usually fairly easily differentiated. An enlarged thymus resembles a Phrygian cap superimposed upon the heart. A dermoid cyst is well anterior and rarely causes displacement of the esophagus or trachea. A paravertebral abscess is easily diagnosed if the possibility is remembered. It is posterior and associated with vertebral destruction. Dilatation of the esophagus due either to stricture or cardiospasm is easily diagnosed with barium but the possibility must be considered.

There are certain causes for increased translucency in lung:

1. Pneumothorax is easily identified, but tension pneumothorax is to be remembered as an important type which may call for intervention.

2. Generalized emphysema in children is usually associated with asthma. When localized it may be conpensatory to collapse or due to the ball valve action of a foreign body. Enlarged mediastinal glands may produce the same effect and the resultant emphysema may be the only indication of tuberculosis.

3. Cysts may be congenital or may result from inflammation such as bronchiectasis.

4. Cavities are seen in lung abscesses and are often indistinguishable from cavities seen in primary tuberculosis. The rare adult type of tuberculosis produces typical tuberculous cavities.

Opacities in the lungs may be divided into several groups:

1. Mottled opacities when localized are usually pneumonia; when generalized are usually miliary tuberculosis, either acute or chronic, though leukemia may produce a similar appearance. Carcinoma metastasizing to lung produces mottled opacities of varying size; in

sarcoma the lesions are usually larger and the appearance is typical.

2. Rounded opacities should be carefully identified as to whether they are pulmonary or pleural. In the lung the commonest cause is tuberculosis. Other causes are rarer, as hydatid cyst, congenital cyst, metastasis, or primary neoplasm. Thoracotomy is usually necessary for diagnosis. In the pleura the usual possibilities are neoplasm, fluid-containing cyst, or encysted effusion. The commonest tumors are those arising from the nervous tissue.

3. Non-rounded opacities in the pleura are almost always clear effusion or pus. Small ones are particularly difficult to recognize and are particularly important as they frequently accompany tuberculosis.

4. Segmental opacities in lung are the most common opacities and represent varying degrees of consolidating or collapse. They are due to inflammation, pressure from tuberculous gland on a bronchus, ulceration of the gland into the bronchus, an aspirated foreign body or primary neoplasm.

5. Linear opacities are accessory fissures, fibrosis, complete collapse of a small segment or bronchiectasis. Fibrosis is accompanied by displacement of mediastinal structures. Bronchiectasis may supervene after collapse due to primary tuberculosis.

An instructive series of roentgenograms accompanies this paper—*E. F. Lang, M.D.*

STEPHENS, H. BRODIE. Congenital pulmonary stenosis. (Editorial) *Surg., Gynec. & Obst.*, June, 1948, *86*, 758–760.

The most frequently encountered type of congenital cardiovascular defect accompanied by cyanosis is termed the tetralogy of Fallot (pulmonary stenosis, dextroposition of the aorta, interventricular septal defect and right ventricular hypertrophy). This malformation results in a greatly diminished flow of blood through the pulmonary artery. Cyanosis and clubbing of the fingers are due to the venous-arterial mixing that occurs through the interventricular septal defect where the flow is from right to left and venous blood from the right ventricle passes out into the overriding aorta directly into the systemic circulation.

Taussig emphasizes certain characteristic clinical findings that are helpful in establishing the diagnosis of the tetralogy of Fallot, to wit, the heart is normal in size, and usually a basal systolic murmur and pure second sound are audible. The electrocardiogram shows a right axis deviation. The roentgenogram demonstrates an absence of the normal fullness of the pulmonary conus and the lung fields are clear. Fluoroscopy shows no pulsations at the hilum of the lungs. There are, however, according to Taussig, many variables in these clinical findings.

Anastomosis of one of the major systemic arteries to the right or left pulmonary artery is often spoken of as the Blalock operation. Blalock and Taussig have advocated this operation to increase the pulmonary blood flow in that there is a definite lack of circulation to the lungs in congenital pulmonary stenosis. The great value of this operation has been demonstrated in several hundred cases that have been managed by Blalock and his associates.—*Mary Frances Vastine, M.D.*

HUGHES, FELIX, A., JR., and WESTPHAL, KEAN F. Amebiasis with pulmonary involvement. *Arch. Surg.*, Sept., 1947, *55*, 304–315.

The authors estimate that about 10 or 20 per cent of the population harbor *Endamoeba histolytica* and suggest that the percentage is at least twice as high in soldiers returning from overseas.

The incidence of hepatic involvement in cases with dysentery apparently varies from about 9 per cent in early well treated cases to 95 per cent in the fatal ones. Pleural and pulmonary complications have been described in 5 to 15 per cent of the patients with dysentery and from 10 to 20 per cent of those with hepatitis.

Once the liver has been involved, the pleura and lungs may become affected as the result of direct extension of the disease through the diaphragm. If the pleura alone is involved, a sterile empyema may develop. Bronchobiliary fistula without empyema or pneumonitis has been described. The most common process, however, is a diffuse pneumonitis of the right lower lobe associated with an abscess or a bronchopulmonary fistula.

The authors believe that amebic hepatopulmonary disease occurs often enough to be included in the differential diagnosis of diseases of the right upper abdominal quadrant or right lower portion of the chest. The diagnosis depends upon the finding of *Endamoeba histolytica* or by the specific response to emetine hydrochloride.

When the pleura becomes involved, cough and pain in the chest aggravated by deep

breathing and finally pleural effusion make their appearance. When the lungs become involved, the cough becomes productive of a bloody purulent sputum in which the organism may often be found.

According to the authors, the earliest roentgen manifestations of threatened bronchopulmonary involvement include elevation and fixation of the right hemidiaphragm plus some irregularity in its configuration. They describe a characteristic triangular shadow in the base of the right lung which extends from the hilum as its apex, to the liver. Bulging with obliteration of the cardiophrenic and anterior costophrenic angles are also described. The presence of a cavity in the lungs and the actual demonstration by means of a contrast substance of the bronchohepatic fistula have been demonstrated.

The records of 3 patients with amebic infections of the lung are reported and their roentgenograms reproduced.—*Philip J. Hodes, M.D.*

Gross, Robert E. Surgical treatment for coarctation of the aorta. (Editorial) *Surg., Gynec. & Obst.*, June, 1948, *86*, 756–758.

This anomaly is more common in men than women. In patients with coarctation who live past the first two years of life, certain generalities can be made. One of four eventualities may overtake him: (1) he may live a long life and have little or no important complaints related to the vascular obstruction; (2) he may die suddenly from aortic rupture. This fatal complication is most common in the third decade of life; (3) he may die from superimposed *Streptococcus viridans* infection, a complication which was most common during the second and third decades. Such a threat is less likely in the present day of chemotherapy; (4) he may die of the hypertensive state—from cardiac failure or from intracranial hemorrhage.

In a series of 40 patients with coarctation treated by the author, the following results were obtained: (These patients ranged in age from seven to thirty-one years.)

1. The thoracic duct was not injured in any case.

2. In 5 instances, only exploration was carried out; the operation was terminated because the narrowed segment of aorta was very long or else the vessels were surrounded by an inflammatory reaction as a result of previous infection.

3. In 35 cases the narrowed portion of aorta was excised and the remaining ends of the vessel were brought together.

4. There were 5 deaths in this last group.

5. Specimens removed from the 35 patients showed a complete block in 5 and in the remainder there was a tiny opening.

6. The optimum ages for procedures of this sort appear to lie between ten and twenty years. —*Mary Frances Vastine, M.D.*

Longmire, William P., Jr. Congenital atresia and tracheoesophageal fistula. *Arch. Surg.*, Sept., 1947, *55*, 330–338.

The author calls attention to the fact that the most important step in the treatment of these patients is the early recognition of the anomaly by the attending physician before the infant's life is seriously threatened by aspiration pneumonia, or dehydration, and starvation.

The success or failure of the operative procedure depends in large part on the general condition of the patient at the time of operation.

The author reports 4 consecutive patients with tracheoesophageal fistulas and esophageal atresia who survived ligation of the fistula and primary anastomosis of the esophagus. All of the patients are now taking feedings by mouth and are gaining weight satisfactorily.—*Philip J. Hodes, M.D.*

Guy, Chester C., and Oleck, Henry T. Traumatic biliary-bronchial fistula. *Arch. Surg.*, Sept., 1947, *55*, 316–329.

The authors call attention to the rather uncommon occurrence of biliary-bronchial fistulas in patients suffering combined thoraco-abdominal wounds. That this is not due to an excessive and early mortality rate is supported by the fact that 70 per cent of patients with abdominothoracic wounds on the right side survive and 50 per cent of the patients with similar wounds on the left side survive. The fact that wounds of the liver are usually either rapidly fatal from hemorrhage or else heal surprisingly well is generally recognized. Of considerable significance also is the fact that even though portal blood and liver commonly harbor pathogenic bacteria, there is no evidence that wounds of the liver show any particular tendency to become infected.

The authors postulate the presence of some interference with the free flow of bile in patients who develop biliary-bronchial fistulas. They also believe that in the presence of considerable

liver necrosis, abscess cavities may result which predisposes to biliary-bronchial fistulas. It is their opinion that the essential pathologic process begins in the liver and that effective surgical therapy must be directed primarily to the liver.—*Philip J. Hodes, M.D.*

ABDOMEN

RITVO, M., and SHAUFFER, I. A. Roentgenographic studies of the gastro-intestinal tract following section of the vagus nerves for peptic ulcer. *New England J. Med.*, April 8, 1948, *238*, 496–501.

Thirty-three patients with bilateral vagus resection for peptic ulcer were studied by film and fluoroscopic methods at the Boston City Hospital. Twenty-nine of the operations were supradiaphragmatic and 4 transabdominal. Thirty patients were male and 3 female. The ages ranged from twenty-one to fifty-seven. Twenty-one patients had no previous gastric surgery, 3 had had previous gastroenterostomies, and 5 had had partial gastrectomy followed by marginal or jejunal ulcer. Of the twenty-one patients without previous surgery, 20 had duodenal ulcers, and 1 had an ulcer on the lesser curvature of the stomach.

In the first weeks after operation, the stomach exhibited dilatation, atonicity and sluggish, ineffective peristalsis. Emptying time was markedly increased. After several months, all of these changes tended to return toward the normal, although one patient with very poor emptying required a subsequent gastroenterostomy. The duodenal bulb improved in caliber after operation. In patients with previous surgery, gastric dilatation and delay was less marked, and the stomal and jejunal ulcers healed promptly.

One patient developed temperature dysphagia after transabdominal vagus resection. Roentgen examination showed changes suggestive of cardiospasm, with narrowing of the lower end of the esophagus, reverse peristalsis and delay in esophageal emptying.—*Henry P. Brean, M.D.*

PEIN, N. K. Neonatal gastrocolic fistula; report of a case. *Lancet*, July 10, 1948, *2*, 53–54.

Report of one case in a five week old infant with greenish slimy undigested food per rectum and a fecal character to vomitus.

Diagnosis proved by roentgenography after a bismuth meal. The bismuth flowed directly from stomach into transverse colon. At operation the fistula appeared to be of inflammatory origin. The baby died two days after operation. At necropsy an ulcer 3 mm. in diameter was found inside the stomach. On section this appeared to be a small chronic gastric ulcer.

The author believes the findings strongly suggest a peptic ulcer which, starting in fetal life, had perforated the adjacent colon.—*J. S. Summers, M.D.*

FALLE, E. DE C. Pyloric spasm simulating congenital hypertrophic stenosis; report of a case. *Lancet*, May 22, 1948, *1*, 794.

A girl, aged six weeks, presented all the classical signs and symptoms of a typical pyloric stenosis and a roentgenographic picture of almost complete obstruction at the pylorus. After conservative medical regimen failed, an exploratory laparotomy revealed no organic pathology. After operation glyceryl trinitrate was used to relax the pylorus. Barium meal study showed a normal appearance.—*J. S. Summers, M.D.*

TAYLOR, H. Carcinoma of the stomach. *Lancet*, April 17, 1948, *1*, 581.

Each year 13,000 people in England die of carcinoma of the stomach. Only 70 per cent of these are seen in a hospital. Five out of 6 are beyond cure when they are first seen in the hospital. Of each 5 hopeless cases, 2 have clinically recognizable metastases on arrival, 1 is generally too ill for operation, and 2 are found to be inoperable on laparotomy. The 1 patient in 6 whose growth is removed submits his life to a 30 per cent risk. If he survives the crisis he has half the normal life expectancy.

The author deplores these facts, then suggests a plan whereby everyone in England under the new health service program and between the ages of forty and sixty-five (roughly 15,000,000 people) may have a barium meal study for even the most vague abdominal complaint. He suggests that if 1 in 25 of this age group had complaints, 50,000 barium meals would be needed each year. This need could be met by ten roentgen-ray units in each of the twelve health service regions, if each unit could do 20 studies a day. This is not an impossible goal.

He is appalled and depressed by the endless succession of hopeless laparotomies or desperate dissections of too extensive growths, and feels

that the proposed program, though not perfect, would increase the number of patients referred for treatment while still in the operable stage.—*J. S. Summers, M.D.*

Kane, F. F. Acute intestinal obstruction in typhoid fever. *Lancet*, July 17, 1948, *2*, 97–98.

This is a report of an unusual case of typhoid fever, admitted to the hospital sixteen hours after onset of symptoms because of history of ingestion of contaminated milk. Combined penicillin and sulfathiazole gave disappointing results. On the evening of the forty-third day after an acute abdominal pain, exploratory laparotomy revealed an acute intestinal obstruction caused by peritoneal bands, probably due to stretching of adhesions formed over areas of typhoid ulceration. Division of the bands relieved the abdominal symptoms, but death followed from typhoid septicemia.—*J. S. Summers, M.D.*

Hansen, Paul Scott. Hemangioma of the small intestine with special reference to intussusception; review of the literature and report of three cases. *Am. J. Clin. Path.*, Jan., 1948, *18*, 14–42.

Hemangiomas (Raiford) compose 6 per cent of benign tumors and 3.4 per cent of all tumors of the small intestine. Fifteen to 30 per cent of all benign tumors of the small intestine cause intussusception, but hemangiomas relatively rarely cause this condition (as will be noted according to the classification). Kaisjer's adaptation of Oberndorff's classification is as follows:

1. Multiple phlebectasia—multiple dilated venous structures, microscopically cavernous in type, grossly pin-head to pea-sized, purple nodules. Rarely cause symptoms.
2. Cavernous hemangioma.
   (a) Diffuse infiltrating cavernous hemangiomas; cavernous tissue usually invades all layers sparing mucosa and muscularis and results in narrowing of the lumen by thickening of the wall. Lesions are probably solitary. Hemorrhage occurs in most. Symptoms of obstruction in 50 per cent.
   (b) Circumscribed often polypoid, cavernous hemangioma. Always single and always cavernous. Not infrequently cause hemorrhage and, when polypoid, can cause intussusception.
3. Simple capillary hemangioma. Capillaries are not grossly dilated as in the foregoing type. Tumor is usually spherical and projects into the lumen and is frequently the size of a plum. Majority are found in the ileum. Hemorrhage, obstruction and intussusception are noted.
4. Hemangiomatosis localized in the gastrointestinal tract. A portion of the widespread hemangiomatosis, involving particularly skin and liver. Relatively asymptomatic, with the exception of occasional hemorrhage, obstruction or intussusception.

Diagnosis of hemangioma of small bowel is rarely made preoperatively. Diagnosis is perhaps improved if the following are noted:

1. Presence of hemangiomas on skin or mucous membrane.
2. The frequent presence demonstrable by roentgen ray of phleboliths, homogeneous or layered in diffuse infiltrating cavernous hemangioma.
3. Significant association of intussusception with polypoid hemangiomas of small intestine.
4. Possibility of roentgen demonstration of indentations and constriction of the small intestinal lumen in cases of otherwise unexplained gastrointestinal hemorrhage.—*Russel R. Jauernig, M.D.*

Colvert, James R. Rectal polyps; diagnosis, five year follow-up and relation to carcinoma of the rectum. *Am. J. M. Sc.*, Jan., 1948, *215*, 24–32.

Rectal polyps rarely present symptoms suggestive of their presence and examination of the patient is incomplete without proctoscopic examination. Patients with rectal polyps should have a double contrast barium enema to rule out polyps higher in the colon.

One cannot be guided by size, shape or general appearance as to whether a polyp is malignant or benign although there is a much higher percentage of ulceration in malignant polyps. Only by prompt removal of a rectal polyp and pathological examination can one determine malignancy or benignity, and thus give patients a maximum chance of permanent cure without major surgery.

Prompt removal of the majority of rectal polyps in this group of cases resulted in a five year cure without any major surgery in 13 out

of 14 patients with malignant rectal polyps.—*Herbert Lobsenz, M.D.*

HARRINGTON, STUART W. Various types of diaphragmatic hernia treated surgically. *Surg., Gynec. & Obst.*, June, 1948, *86*, 735–755.

The reason for the different types of diaphragmatic hernias is the unusual embryologic formation of the diaphragm which makes for weak parts through which these hernias may occur.

Nontraumatic Hernia. A nontraumatic diaphragmatic hernia may be congenital or acquired. If it is congenital, the hernia is attributable to embryologic deficiency and usually does not have a hernial sac. The common sites of a congenital hernia, in the probable order of frequency of occurrence, are:

1. Through the pleuroperitoneal hiatus. These hernias occur in the posterolateral portion of the diaphragm and are due to failure of fusion of the pleuroperitoneal membrane and the septum transversum. The defect is usually triangular with the apex toward the median portion of the diaphragm. These hernias do not have hernial sacs and there is a direct communication between the abdominal and the thoracic cavity.

2. Through a gap left by partial absence of the diaphragm, a gap which is usually situated in the posterior portion of the muscle. These hernias may be considered an enlargement of the foregoing pleuroperitoneal type in that the essential difference is a much more extensive congenital defect in the formation of the diaphragm. There are more abdominal viscera involved than in the pleuroperitoneal hernia as the stomach and spleen, also the large and small intestine are often included.

3. Through the esophageal hiatus due to a deficiency of the circular muscle bundles of the hiatus.

4. Through the esophageal hiatus due to deficiency of the esophagus which is not elongated sufficiently to extend to the diaphragm thus causing varying amounts of stomach to remain above the diaphragm depending on the amount of shortening of the esophagus.

5. Through an anterior subcostosternal opening (foramen of Morgagni or Larrey's spaces). This is one of the two types of diaphragmatic hernia which have a hernial sac. The other type is that through the esophageal hiatus. These subcostosternal hernias are essentially direct hernias through a congenital defect in the structure of the diaphragm or faulty attachment of the diaphragm to the sternum and costal cartilages.

If the hernia is acquired after birth, the sites of occurrence are: (1) through the esophageal hiatus, a type in which there is a hernial sac, (2) through the region of fusion of the anlage of the diaphragm, and (3) at sites named under the congenital types of hernia.

Traumatic Hernia. Traumatic diaphragmatic hernia may be caused by direct or indirect injury or by inflammatory necrosis of the diaphragm. In cases of indirect injury of the diaphragm, the hernia may occur at any point, including points of embryologic fusion, but the most common sites are the dome and the posterior half of the left part of the diaphragm.

Rupture of the diaphragm may be the result of inflammatory necrosis, which, in turn, has been caused by subdiaphragmatic abscess. Again, rupture may follow necrosis caused by drainage tubes which have been introduced into empyema cavities.—*Mary Frances Vastine, M.D.*

MEYER, KARL A., ROSI, PETER A., and STEIN, I. F. Studies on vagotomy in the treatment of peptic ulcer. II. Clinical evaluation. *Surg. Gynec. & Obst.*, May, 1948, *86*, 524–529.

Thirty-five patients with peptic ulcer have had vagus section and have been followed clinically from six to fifteen months. Complete vagotomy causes immediate cessation of peptic ulcer distress with apparent healing of the ulcer. There are marked changes in gastric secretory and motor mechanisms following complete vagotomy. It is not certain to what extent the altered gastric function is permanent. Little is known of the effect of vagotomy on the other organs innervated by the vagi.

It is still too early to make a final evaluation of vagotomy in the treatment of peptic ulcer. Until more information is available concerning the incidence of recurrence of ulcer distress and the possible late sequelae, the use of vagotomy should be limited to clinical investigation, with one exception. Complete vagotomy is the method of choice in the treatment of marginal ulcer following gastric resection.—*Mary Frances Vastine, M.D.*

Stein, I. F., and Meyer, Karl A. Studies on vagotomy in the treatment of peptic ulcer. III. Physiological aspect. *Surg., Gynec. & Obst.*, Aug., 1948, *87*, 188–195.

Following complete vagotomy there is a marked reduction of the night secretion and the basal secretion. The secretory response of the stomach to caffeine and histamine is greatly reduced.

Insulin hypoglycemia produces an increase in gastric secretion and usually motility. This action is abolished by complete vagotomy. There are no spontaneous hunger contractions in the fundus of the stomach up to nine months following complete vagotomy.

The vagi are the sole mediator of the cephalic phase of gastric secretion. They are the most important factor concerned in the interdigestive period and a contributing factor in the gastric phase of secretion. Complete vagus section in some manner interrupts a mechanism necessary for chronicity of peptic ulceration.—*Mary Frances Vastine, M.D.*

Berg, Benjamin N. Gastric ulcers produced experimentally by vascular ligation. *Arch. Surg.*, Jan., 1947, *54*, 58–66.

In the past, attempts have been made at an experimental level to prove Virchow's hypothesis that human gastric ulcers were of vascular origin. For the most part, no significant results were obtained which was attributed to the fact that the experimental animal, dogs, had a highly efficient collateral circulation in the region of the stomach.

Rats had not been previously used for this experiment. They seemed ideally suited for it because the main gastric vessels do not communicate directly with one another as they do in dogs and in human beings.

Three groups of experiments were carried out, one in which the antral blood supply was ligated, a second in which unilateral ligation of the fundic vessels was accomplished and a third in which bilateral ligation of the fundic vessels was carried out.

Ten of the 18 rats in which the antral blood supply had been ligated developed punched-out penetrating ulcers. In contrast, when unilateral ligation of the fundic vessels was established, only 1 of the 12 animals so ligated revealed a ulcer. When bilateral ligation of the fundic vessels was established, 5 of the 8 animals used revealed similar ulcers. It was noteworthy that despite extensive gastric ulcerations, all the rats kept for periods of eight days or sixteen days survived ligation.

This suggests that a separate factor is concerned with involvement of the muscular coats of the stomach after the initial break of the mucosa has taken place.—*Philip J. Hodes, M.D.*

Ferrer, José M., Jr. The effect of tetra-ethyl ammonium chloride on gastric secretion and acidity in peptic ulcer. *Surg., Gynec. & Obst.*, July, 1948, *87*, 76–78.

The excessive secretion of acid gastric juice at night when the stomach is empty of food has been shown by Dragstedt to be a major contributing factor in the origin and particularly in the continuing activity of peptic ulcers. He has further shown that the amount of free stomach acid and the volume of the night gastric secretion are excessive in patients with peptic ulcer as compared with normal persons or patients with other diseases. To decrease this night secretion, Dragstedt devised an efficient method of sectioning the vagus nerves to the stomach. Following complete section of both vagus nerves, a marked decrease in the secretion of acid gastric juice has been uniformly observed. Unfortunately, however, the effects of vagus section have not been entirely beneficial. Many observers have reported postoperative gastric atony, dysphagia, bouts of diarrhea, etc.

Because of the unfortunate effects of vagus section the author conceived a method of temporary vagal block to effectively decrease the excessive night gastric secretion in the patient with peptic ulcer without interfering with vagal function during the day when acid gastric juice and peristaltic activity are needed in the processes of digestion.

Conclusions

1. Tetra-ethyl ammonium chloride (etamon) prevented excessive night gastric secretion and acidity in 12 out of 17 tests of patients with peptic ulcer.

2. Intramuscular etamon dosage of approximately 20 milligrams per kilogram of body weight is usually adequate to produce the desired effect if repeated at four hour intervals.—*Mary Frances Vastine, M.D.*

METZ, ARTHUR R. Duodenal regurgitation. *Arch. Surg.*, Sept., 1947, *55*, 239–245.

The author believes that duodenal regurgitation has not been given proper consideration as the cause for symptoms in recent years.

The symptoms vary with the degree of obstruction. When the obstruction is minimal recurring attacks of nausea with some disturbed appetite are common. With progression the nausea increases and is frequently attended by a sense of fullness in the right upper abdomen. Vomiting and ultimately considerable loss of weight ensue.

The diagnosis depends upon the demonstration by roentgen methods of a dilated duodenum associated with to and fro regurgitation of the barium which seems to stop abruptly in the region of the spine. The author believes that it is pressure by the mesenteric vessels on the duodenum where it passes over the spine in ptotic individuals that is responsible for the duodenal regurgitation.

The author has operated upon 14 patients with relief of symptoms. He believes the surgical treatment of choice following careful medical management is a duodenojejunostomy.—*Philip J. Hodes, M.D.*

HEIFETZ, CARL J., and SENTURIA, H. R. Acute pneumocholecystitis. *Surg., Gynec. & Obst.*, April, 1948, *86*, 424–433.

By acute pneumocholecystitis is meant an acute infection of the gallbladder characterized by the production of gas within the gallbladder lumen. The diagnosis is made roentgenographically, the gallbladder being visualized through the contrast medium of gas. That gas-forming organisms are frequent inhabitants of the biliary tract has been known for years. While a majority of normal and diseased gallbladders are sterile, others contain a wide variety of bacterial flora, headed in frequency by the *Escherichia coli communis* and the *Clostridium perfringens*. Besides these organisms other gas-formers, often difficult to classify, may exist in the bile tract. For the most part they seem to exist as harmless saprophytic inhabitants.

In evaluating the significance of the role of gas in acute pneumocholecystitis, certain considerations of etiological importance in ordinary cases of acute cholecystitis should be discussed. There is now rather universal agreement that a prime etiologic factor is obstruction at the out let of the gallbladder. Berk reported that blockage of the outlet of the gallbladder by calculi was found in over 92 per cent of the cases of acute cholecystitis collected from the literature.

It is likely that the infections of the gallbladder which produce gas are rarely associated with *Clostridium perfringens*, but instead with other gas-forming organisms. Another inference is that the development of acute cholecystitis in these cases occurs independent of the bacteria present, and the formation of gas is a rare complication which depends upon the presence of a gas-former within the gallbladder at the time, and, more important, upon conditions enhancing its gas-producing potentialities. That these poorly understood conditions are important can be adduced from the fact that many acute gallbladders contain *Clostridium perfringens* and other gas-forming organisms without producing gas.

Summary

1. Eight cases of acute pneumocholecystitis have been collected from the literature and reviewed. Two additional cases diagnosed preoperatively are added to the literature.

2. By pneumocholecystitis is meant acute infection of the gallbladder characterized by the production of gas within the gallbladder. Pericholecystic infiltration with gas may or may not be present.

3. Necrosis of the gallbladder with perforation is a frequent finding. However, the virulence of the infection is relatively less than in gas bacillus infections elsewhere in the body.

4. Bacteriologically, no constant distinctive organisms are demonstrable.

5. Surgical therapy alone is effective since the gallbladder wall is subject to greater tension when gas is present and there is a consequent greater risk of gangrene and perforation.—*Mary Frances Vastine, M.D.*

KITTLE, C. FREDERICK, JENKINS, HILGER P., and DRAGSTEDT, LESTER R. Patent omphalomesenteric duct and its relation to the diverticulum of Meckel. *Arch. Surg.*, Jan., 1947, *54*, 10–36.

This is a comprehensive and clearly written review of the history, embryology, anatomy and pathology of patent omphalomesenteric duct and the diverticulum of Meckel. In addition, the authors summarize the findings in 102 cases of patent omphalomesenteric duct reported in the literature and 26 additional cases in which prolapse of the intestine occurred through this structure.

According to the authors, Meckel's diverticulum and the patent omphalomesenteric duct are the consequence of the same defect in embryologic development, differing only in the extent to which the omphalomesenteric duct fails to obliterate. Whereas Meckel's diverticulum occurs in about 1 to 2 per cent, there were but 2 patients with patent omphalomesenteric ducts among the 30,000 births at the Chicago Lying-In Hospital of the University of Chicago during a ten year period.

Usually the defect is first noted at birth when a small red mass is seen at the umbilicus after the cord structures have sloughed away. The presence of fecal drainage is almost pathognomonic. The authors discourage the use of a probe because of the possibility of perforating the bowel and advise the introduction of radiopaque material through a small catheter in order to diagnose or exclude the congenital defect. The presence of a short communication which extends from the anterior abdominal wall down to the ileum is pathognomonic. The variant is three to four times as common in males as in females.

The patent ompthalomesenteric duct must be distinguished from granulation tissue in the stump of the umbilical stalk due to infection. A patent urachus with or without urinary discharge may be confused with it. In rare instances, a ligated loop of bowel which may have prolapsed into the umbilical cord may simulate the patent duct.

Operation for excision of the patent duct is advised as soon as the diagnosis is made in order to avoid the hazard of intestinal prolapse. When this complication occurs, the prognosis becomes increasingly unfavorable depending upon the duration of the prolapse.

This article contains a complete bibliography and of particular interest is the diagrammatic illustration of the different variations in the development of the omphalomesenteric duct which is included.—*Philip J. Hodes, M.D.*

Beneventi, F. A., and Twinem, F. P. Neuroblastoma. *J. Urol.*, Aug., 1948, *60*, 235–241.

Neuroblastoma is next in frequency to Wilms' tumor as the most common abdominal tumor in infancy and childhood.

The authors report the case of a twenty month old child who had developed an abdominal enlargement within two weeks following hospital discharge for an otitis media. This swelling increased rapidly and cervical nodes became palpable at the same time. On admission to the hospital the child had a fever of 101° F. and a hard palpable mass in the left side of the abdomen, extending from the costal border to the pelvis and well across the midline. Excretory urograms showed a normal right kidney and no demonstrable function on the left. A diagnosis of Wilms' tumor was made. At operation the left kidney could not be identified and the tumor mass was not removable. The patient died the day following operation. At postmortem examination the left kidney was incorporated in a huge tumor mass. The kidney pelvis was compressed but not invaded. The left adrenal gland could not be identified. The histopathological diagnosis was neuroblastoma.

Neuroblastoma is an extremely malignant, rapidly growing tumor, made up of undifferentiated neuroblasts. Virchow first separated these tumors from the sarcoma group and called them gliomas. Around 1900 the group was divided into Hutchinson and Pepper types, the former arising in the left adrenal and metastasizing to the skull and the latter arising in the right adrenal and metastasizing to the liver. These types are now felt to be unjustifiably separated. Wright, in 1910, first used the term neuroblastoma after Kuster had described the rosette formation of the cells in 1905.

Neuroblastoma can arise from the adrenal medulla or from sympathetic nervous tissue of other structures.

The clinical picture is that of a pale underweight infant or young child with a rapidly enlarging abdomen. In the differential diagnosis Wilms' tumor is the most difficult problem. Bone involvement in Wilms' tumor is rare and common in neuroblastoma. Neuroblastoma is more resistant to roentgen therapy.

Other lesions which may occasionally cause difficulty in the differential diagnosis include scurvy, adrenal cortical tumors, lymphosarcoma, ovarian tumors, leukemia and Ewing's sarcoma.

Farber, who has reported a series of 40 cases, believes that every solid tumor in infancy should be considered malignant until proved otherwise by pathological examination, and that the tumor should be removed within forty-eight hours with no delay for preoperative roentgen therapy. He recommended post operative roentgen therapy. Farber reported 25 per cent survival of from three to eight years. He also observed that these tumors may undergo spontaneous hemorrhage and necrosis

and disappear without any therapy other than biopsy. He also observed that these tumors may spontaneously mature into benign ganglioneuromas.—*Rolfe M. Harvey, M.D.*

MILLER, EDWIN. Malrotation of the colon. (Editorial) *Surg., Gynec. & Obst.*, Aug., 1948, *87*, 235–237.

The term "malrotation," strictly speaking, is that state of affairs that is present when either the normal counterclockwise rotation or the abnormal clockwise rotation of the midgut has been arrested before it is completed and one or more segments of bowel, usually the cecum, has become fixed by adhesions in an abnormal position.

From a clinical standpoint the end result is almost always an acute or chronic obstruction of the duodenum. Occasionally the onset of trouble is delayed for weeks, months or even years and occasionally the onset is insidious, but in great the majority of cases the picture is acute and comes on a few days after birth. The outstanding feature, of course, is the persistent vomiting of bile and it is usually associated with fullness in the epigastrium and flatness of the lower belly. If, however, the obstruction is incomplete and intermittent in character, the several loops of twisted midgut may contain much fluid and gas and a mass may be readily palpable.

Early surgery is the key to success and it consists essentially in untwisting a volvulus or completely severing abnormal adhesions associated with a malrotated colon. Through an ample incision the entire abdomen is explored. The distended duodenum is noticed at first glance and the absence of the colon in its normal place gives one an immediate clue as to the basic underlying pathology.—*Mary Frances Vastine, M.D.*

POTTS, WILLIS J. Congenital atresia of intestine and colon. (Editorial) *Surg., Gynec. & Obst.*, April, 1948, *86*, 504–506.

Atresia of the bowel is a rare congenital deformity which cannot be diagnosed until symptoms of obstruction appear. Vomiting is the first symptom. If the obstruction is high, persistent vomiting will begin shortly after birth; if the obstruction is near the ileocecal valve, the most common site, vomiting may not become alarming until the second or even the third day of life. Most atresias of the bowel are distal to the ampulla of Vater; the vomitus therefore is green. A newborn baby who vomits green material has intestinal obstruction until proved otherwise. It does not have pyloric stenosis because the vomitus of a baby with pyloric stenosis is never green.

Distention of the abdomen varies with the level of obstruction. One should not be led astray by finding a scaphoid abdomen in these patients. Progressive and finally marked distention of the abdomen, however, typifies atresias of ileum or colon.

The difference between a normal meconium stool and that of an infant with intestinal atresia is not great enough to be impressive.

The suspected diagnosis of atresia of the intestine or colon is easily confirmed by examination of roentgenograms made of the baby in the upright position. Widely distended loops of bowel showing fluid levels will be seen. The number of distended loops of bowel and their position in the abdomen will indicate the level of obstruction. These babies should not be given barium; its use is unnecessary for diagnosis, it tends to clog the intestine and if aspirated produces tracheobronchitis.

The treatment of congenital atresia of the intestine and colon is surgical.—*Mary Frances Vastine, M.D.*

SARNOFF, STANLEY J., ARROWOOD, JULIA G., and CHAPMAN, WILLIAM P. Differential spinal block; the investigation of intestinal dyskinesia, colonic atony, and visceral afferent fibers. *Surg., Gynec. & Obst.*, May, 1948, *86*, 571–581.

All patients studied were from the surgical services of the Massachusetts General Hospital. Five patients were given a differential spinal block in an attempt to alter temporarily bowel function. Two additional patients had the visceral afferent pathways (carrying the sensation of distention) tested while under the influence of a high differential block.

Conclusions

1. Visceromotor fibers to the intestinal tract are of about the same size and degree of myelination as other sympathetic efferent fibers insofar as this similarity is indicated by their relative susceptibility to procaine hydrochloride.
2. Block of sympathetic efferent fibers to the intestinal tract was followed by a definite increase in propulsive bowel activity and co-

ordination of transport in 3 of the 5 patients studied, and a probable alteration in bowel activity in the remainder.

3. Visceral afferent fibers subserving the sensation of distention are relatively large, myelinated axones insofar as this is indicated by their relative refractoriness to procaine hydrochloride. They are not blocked by that concentration capable of interrupting sympathetic efferents (vasomotor, sudomotor and visceromotor) and fibers carrying the appreciation of pinprick.

4. Impulses traveling over autonomic nerves can contribute significantly to the production of a syndrome consisting of distention, obstipation, tympanites, fecal vomiting, and cramplike abdominal pain which can be easily confused with acute, mechanical, intestinal obstruction. This type of neurogenic obstruction may be sufficiently obscure to precipitate numerous abdominal interventions and the patient finally falls into that category labelled "the multiple laparotomy syndrome."

5. The conventional type of full spinal block and a differential spinal block are of value in relieving this type of bowel obstruction, and in helping to establish the diagnosis. The differential spinal block is more specific as an investigative tool inasmuch as the fibers carrying distention pain are not interrupted. The alteration of bowel activity is therefore presumably due to a block of visceromotor fibers. It has the added advantage of allowing the patient to use her abdominal musculature in expelling colon contents at the peak of intestinal activity.

6. Impulses arising in the autonomic nervous system may be important in colonic atony as seen in the adult, since block of the visceral efferents can greatly alter colon activity in such a patient.

7. It is of great importance to withhold medication when attempting to augment intestinal activity and transport by means of spinal anesthesia.—*Mary Frances Vastine, M.D.*

Leithauser, D. J. Atypical adynamic ileus apparently caused by nutritional (thiamine chloride) deficiency. *Surg., Gynec. & Obst.*, May, 1948, *86*, 543–550.

The cases of 6 patients with adynamic ileus with severe abdominal distention are reported to call attention to the fact that symptoms suggesting the presence of intestinal obstruction may be caused by thiamine chloride deficiency. In these cases, the distention was not controlled by mechanical decompression or administration of prostigmine but responded dramatically to administration of thiamine chloride and vitamin B complex.

In the first case in which the condition was recognized as resulting from vitamin deficiency, the patient had a sore mouth which directed attention to this possibility. Specific questions brought to light a history of chronic alcoholism. In this instance, a preoperative diagnosis of "annular carcinoma of the sigmoid" had been made on the basis of obstructive symptoms and the roentgenographic findings but no such lesion was found at operation. However, the abdominal distention increased after operation and was not brought under control until vitamin therapy was instituted. The second patient, also an alcoholic, was subjected to celiotomy for "acute appendicitis," but at operation there was so much intestinal distention that the appendix was not readily accessible and was not removed. When uncontrollable distention continued after operation the experience in the first case was recalled and vitamin therapy was instituted, with equally striking results.

The author suggests that nutritional deficiency should be suspected and a therapeutic trial of vitamins made in cases of abdominal distention in which the evidence does not justify a positive diagnosis of mechanical obstruction of the intestines. He suggests also that thiamine chloride should be administered at the time of operation to prevent postoperative distention, especially in patients whose nutritional status is at all questionable or in whom there is any suspicion of liver damage.

It is the writer's routine practice to administer parenterally vitamin B complex with high thiamine content to all surgical patients the day before operation, and daily for three days after operation. Since this has been done, postoperative distention has been almost entirely eliminated.—*Mary Frances Vastine, M.D.*

GENITOURINARY SYSTEM

Antopol, W., and Goldman, Lester. Subepithelial hemorrhage of renal pelvis simulating neoplasm. *Urol. & Cutan. Rev.*, April, 1948, *52*, 189–195.

Seven cases with hematuria are presented in which pyelograms showed filling defects in the kidney pelvis suggesting renal neoplasm. The right kidney was involved in 6 of these cases.

In the extirpated specimen the pelvic defects

were found to be due to subepithelial hemorrhage of the renal pelvis. Superficial cortical infarctions were present in 5 of the cases. The subepithelial hemorrhage is obviously due to vascular injury. There was definite history of trauma in only 2 of the cases. The authors feel that because the pelvis and hilar blood vessels are poorly supported in the sinus of the kidney it might require only minimal trauma, even the inertia of the kidney, due to sudden changes of position or motion, to produce this lesion. This view may be supported by the fact that 6 of these 7 cases were found in the more mobile right kidney. Four of the cases also exhibited congenital malformations consisting of (1) extrarenal pelvis, (2) faulty ureteral insertion, (3) bifid pelvis and double ureter and (4) aberrant vessels with extensive fetal lobulation. The authors feel that these congenital abnormalities might predispose to or exaggerate insults to the organ.

The cases are difficult to evaluate by single roentgen examination. Since pelvic hemorrhage may be absorbed, a later examination may show regression to normal. The authors believe that retrograde pyelograms should be taken after partial filling due to the rigidity and inelasticity of these pelves.—*William G. Johnson, M.D.*

HALE, NATHAN G. Primary papillary carcinoma of the ureter; report of 2 cases. *Urol. & Cutan. Rev.*, Jan., 1948, *52*, 3–7.

The author presents 2 cases of primary ureteral papillary carcinoma along with an excellent historical review and discussion of the lesion. In 1936 Rusche and Bacon concluded there were 93 cases of proved primary carcinoma of the ureter. Review of the literature to date reveals 198 cases. The majority of cases are between the ages of sixty and eighty.

The diagnosis of this lesion is usually made at operation. Only 7 cases have been diagnosed preoperatively, the usual diagnosis being functionless kidney or non-opaque calculus. There was no dye shadow found on the affected side in one-half of the 18 cases reported by Cook and Counseller in 1941. In 35 per cent of their cases the ureteral tumor was visible at the bladder orifice. A suggested aid to diagnosis is the passage of a small catheter and taking serial ureterograms at various levels. The increase of reports of this lesion in the past decade suggests improved diagnostic methods better applied.

All treatment has been disappointing. Only 1 patient survived over five years in the 45 cases reviewed in the past nine years. Metastases are widespread due to the abundant lymphatic supply of the ureter. Metastasis has been reported in lungs, liver, bones, bladder, lymph nodes and female genital organs. In 25 per cent of the cases implants have been found in the bladder either before or after diagnosis.

Nephro-ureterectomy with removal of the bladder cuff in one stage is the operation of choice. Neither of the 2 cases reported by the author had the bladder cuff removed. Roentgen therapy as a postoperative adjunct is questioned although the author states his knowledge of its real worth is meager.—*William G. Johnson, M.D.*

MELICK, W. F., and VITT, A. E. The present status of aortography. *J. Urol.*, Aug., 1948, *60*, 321–333.

The authors summarize the literature dealing with early attempts to visualize the circulatory tree. The original reports in experimental animals were unfavorable because of high mortality rates. Fariñas made aortograms by passing a catheter up the femoral artery and injecting 70 per cent diodrast. In 1942 the translumbar method of injection was introduced. The authors have not found one fatality in over three thousand reported injections using this method.

The technique of the translumber route consists of insertion of the needle just below the twelfth rib, four fingerbreadths to the left of the spine. The needle is directed medially and superiorly until the vertebral body is encountered and then withdrawn slightly and directed laterally just past the body into the aorta. The authors state that there are no other important structures which might be hit accidentally. Occasionally, in arteriosclerotic individuals, bleeding may result from strking an atheromatous plaque. A pressure system, rather than hand injection, is used. An ideal medium has not been found. Seventy per cent diodrast does not outline smaller arteries well. The medium employed by the authors is 80 pei cent sodium iodide. Iodism and allergic reactions have occurred.

Aortography should not be employed in the presence of severe liver damage, nephritis, uremia, or in the presence of iodine sensitivity. The authors test for sensitivity by injecting a few drops of the medium intravenously and they routinely give 1,000 cc. of normal saline containing 500 mg. of vitamin C intrave-

nously following aortography. The injection is performed under general anesthesia. A high speed Bucky and a 500 ma. capacity tube are required for short exposures.

The particular value of aortography is in the following groups of cases:

1. Ureteral blockage associated with nonfunctioning kidneys.
2. Extraperitoneal tumors.
3. Renal hematuria with a normal pyelogram.
4. Renal hematuria with a deformed pyelogram.
5. Renal tumors to determine operability.
6. Hydronephrosis due to aberrant vessels.
7. Renal anomalies to determine the length of the renal pedicle.
8. Hypertension to show renal blood flow and relative renal mass.
9. Renal cysts.
10. Placental visualization in pregnancy.—*Rolfe M. Harvey, M.D.*

Edelstein, J. M., and Marcus, S. M. Primary benign neoplasm of the ureter. *J. Urol.*, Sept., 1948, *60*, 409–417.

The authors summarize the cases of benign neoplasms of the ureter which have been reported. They found a total of 27 verified cases and rejected an additional 16 cases in which the diagnosis was not verified.

They report the case of a sixty year old white male who had complained of hematuria for three years. A ureteropyelogram showed a large filling defect in the left mid-ureter, 2 cm. in diameter. The ureter was removed together with the left kidney. A papilliferous tumor mass was found in the ureter and a histopathological diagnosis of transitional type papilloma was made.

The commonest symptoms of ureteral tumors are hematuria, which occurs in 75 per cent of cases, pain and enlargement of the kidney on the affected side. A ureterogram is essential for the diagnosis. Malignancy, tuberculosis, calculi and strictures must be ruled out. Ureteronephrectomy is the accepted treatment.

The authors classify benign tumors of the ureter as (A) epithelial: papilloma; and (B) non-epithelial: myoma, lipoma, fibroma, hemangioma, etc.—*Rolfe M. Harvey, M.D.*

Campbell, Meredith F. Congenital hydronephrosis and hydroureter. (Editorial) *Surg., Gynec. & Obst.*, Aug., 1948, *87*, 237–239.

About 12 per cent of all individuals are born with some variety of urogenital tract malformation and of these approximately one in six has congenital obstruction in the upper urinary tract. Renal excretion begins between the fifth and sixth month of fetal life and for this reason, when obstruction exists, advanced hydronephrosis and other urinary backpressure damage often exists at birth.

Stricture is by far the commonest congenital urinary obstruction. This includes congenital contracture of the vesical outlet. Aberrant renal vessels sometimes compress the ureter. Occasionally the ureter is compressed by congenital bands or ureteral diverticulum.

Congenital ureteral stricture occurs most often at the pelvic junction. This, as well as congenital stricture in other body systems such as the biliary and intestinal tracts, is simply an anomalous narrowing of the lumen. These strictures usually involve only a short section of the ureter. In some cases only renal exploration will demonstrate whether the obstruction is due to stricture, aberrant vessel blockage at the pelvic outlet, ureteral compression by fibrous bands, enlarged lymph nodes or purulent collections.

Stricture below the ureteropelvic level occurs predominantly at the ureterovesical junction where it is bilateral in about a third of the cases. As a rule, the dilation is greatest just above the stricture and this often acts as a buffer to spare the kidney the full harmful effects of the urinary backpressure.

About 25 per cent of all kidneys have an anomalous blood supply with aberrant arteries, veins or both passing from the upper pole, laterally from the cortex, or downward or upward and medially from the lower pole. The last group often cause obstruction of the upper ureter.—*Mary Frances Vastine, M.D.*

McGavran, H. G. Giant prostate without symptoms: neurofibroma. *J. Urol.*, Aug., 1948, *60*, 254–259.

The author reports the case of a giant prostate because of its extreme size, the fact that the tumor was not an adenoma, the fact that the tumor did not produce symptoms of prostatism, and finally because erosion of the pubis resulted from the huge size of the tumor.

The patient was a forty-four year old male who had had a five year history of right lower back pain radiating down the right leg. Roentgenograms of the back and hip were repeatedly negative. There were no urinary symptoms.

By rectal examination a smooth tumor mass was felt, too large to permit passage of the finger beyond it. The mass could be palpated abdominally three fingers above the symphysis.

A cystourethrogram showed marked displacement of the bladder upward and to the left. There was a marked indentation on the bladder wall from tumor pressure. The body and superior ramus of the right pubic bone was almost completely eroded. At operation a huge prostatic tumor was removed. A small bone sequestrum came out with the tumor. Postoperative roentgenograms showed gradual ossification of the os pubis following postoperative roentgen therapy, which was given for nine days (dosage not stated). The pathological report was neurofibroma. The tumor weighed 839.5 grams. This is the second largest benign tumor of the prostate which has been reported.—*Rolfe M. Harvey, M.D.*

BEATTY, R. P. Life expectancy in carcinoma of prostate; a five year survey of eighty-eight cases. *J. Urol.*, Aug., 1948, *60*, 264–268.

This is a supplementary report on 20 additional cases of prostatic cancer, the author having previously reported 68 cases. The diagnosis was made, when biopsy was not secured, on the presence of weight loss, anemia, elevation of the serum acid phosphatase, presence of a fixed hard prostate by rectal examination and roentgen evidence of bone metastases.

Fourteen cases of the recent series had roentgenograms of the spine and pelvis, but only 4 showed evidence of metastases, in spite of the fact that 12 had elevation of the serum acid phosphatase. Of the entire series of 88 cases, 35 are still living and 26 are known to be dead. The average survival rate of the cases which have been followed is 2.73 years for the known dead cases, and two years for those who are still alive.—*Rolfe M. Harvey, M.D.*

SKELETAL SYSTEM

HAUSER, EMIL D. W., and CONSTANT, GEORGE A. Skeletal hemangio-endothelioma; a case report. *J. Bone & Joint Surg.*, April, 1948, *30A*, 517-521.

A case of skeletal hemangio-endothelioma in a white woman, forty years old, is reported which demonstrates some of the complications encountered in diagnosis and treatment of the condition. The patient is still living, nine years after the diagnosis was confirmed. A single lesion was shown in each hip and the question arose as to whether there are two primary lesions, or whether one was a metastasis. The history and course of the case led to the opinion that there were two primary lesions.

Roentgenograms revealed a cystic lesion in the neck of each femur with an ununited pathological fracture, sustained one year previous to admission to the hospital, in the right. Serial studies later showed increased destruction of both femurs with invasion of the surrounding soft parts.

The authors believe that any skeletal lesion, which appears in the roentgenogram as cystic, trabeculated, with invasion of the surrounding soft parts, merits the consideration of skeletal hemangio-endothelioma in the differential diagnosis. Surgical intervention is the only procedure that at present offers any benefit. Roentgen therapy did not resolve the lesion.

Nine years after operation the tumor had increased in size and had invaded the entire pelvis. In spite of the enormous size of the tumor and the pathological fracture of the right femur, the patient is able occasionally to be up in a wheel chair. No metastases were found when the patient was last examined.—*R. S. Bromer, M.D.*

FAIRBANK, H. A. THOMAS. Osteopetrosis; osteopetrosis generalisata, marble bones; Albers-Schönberg's disease, osteosclerosis fragilitas generalisata. *J. Bone & Joint Surg.*, May, 1948, *30B*, 339–356.

This paper includes a short description of the familial and hereditary tendencies, the sex incidence, age of occurrence and etiology of osteopetrosis. Included also, at somewhat greater length, are discussions of the clinical features, roentgenographic features and pathological changes of the disease with a paragraph on the diagnosis from a differential standpoint. Seven cases are described with numerous reproductions of the roentgen findings.

A distinct familial tendency is found and the disease is occasionally inherited. It affects both sexes and is found at all ages from fetal life to seventy-five years. It undoubtedly begins before birth in some cases and possibly in most. The cause is unknown. The chief features of the syndrome are abnormal density of the bones with or without fragility, a strong tendency to anemia which may be severe and fatal, and optic atrophy. In some cases there is a tendency

to fracture, but, on the whole, fragility of the bones has been much exaggerated. When they occur, fractures are sharp, abrupt and transverse. The anemia results from reduction of the blood-forming marrow in the sclerosed bones and eventually becomes of the aplastic type. Cases in which it was leukemic and even apparently pernicious in type have been described.

As seen in roentgenograms, the extent to which bones are affected varies considerably. The whole of a bone including the epiphyses may be uniformly dense and completely structureless. Fine striations, both transverse and longitudinal, may be seen in the metaphyses and epiphyses. Occasionally less dense areas may be seen in the sclerosed bone. The parts unaffected by petrosis may show osteoporosis but this is sometimes more apparent than real. As a rule, the metaphyses are more affected than the shafts of the long bones; they are commonly enlarged or clubbed, the enlargement ending abruptly at its junction with the diaphysis. Occasionally the reverse holds good, the diaphysis being more affected, with the metaphyses less dense and either normal or clubbed. A streaky or patchy arrangement of the denser bone is very rarely seen in cases of generalized osteopetrosis. Apart from the clubbing, the shape of the bones in both varieties is unaffected. In some cases a narrow clear transverse band marks the junction of the diaphysis and the enlarged metaphysis. If, in a child, the tendency to form petrosed bone ceases suddenly after a time, the epiphyses will show a dense center surrounded by a halo of normal density. The ilia often show alternating dense and clear curved bands parallel to the crests. The carpal and tarsal bones, although sometimes universally dense or mottled, frequently show a dense center surrounded by a halo of clearer bone; or the reverse may be seen when the pathological change began later than usual. As a rule the phalanges are decidedly less affected than the rest of the skeleton and they show a dense transverse band of varying width in the metaphysis, close to, or a short distance from, the epiphyseal lines. The skull shows density which is particularly marked at the base. The pituitary fossa is usually rather small, and the posterior clinoid processes are thick and clubbed, almost closing in the fossa. The air sinuses are invisible and the frontal and nasal bones may be thickened as well as dense. The maxillae may be affected but the mandible almost invariably escapes. Calcification of the dural folds may be seen. The vertebral bodies, if not uniformly dense, usually present two horizontal bands of dense bone above and below, with a less dense band between. The ribs are affected, sometimes irregularly, but the rib cartilages escape.

The diagnosis is usually easy when the roentgen examination has once been decided upon. In mild cases, chronic poisoning with lead or phosphorus must be excluded, while in certain localities, fluorosis is worthy of consideration. Juxta-epiphyseal bands of increased density in the metaphyses have been reported in cretins which disappeared under treatment. Occasionally, limited distribution of the dense bone may give rise to difficulty in classification; such cases are probably best placed according to Fairbank, in the melorheostosis group.

The roentgenograms of the reported cases are well worth study.—*R. S. Bromer, M.D.*

Fejer, E. Über einen interessanten Fall von Melorheostosis. (An interesting case of melorheostosis.) *Acta radiol.*, 1948, *29*, 112–116.

A case of melorheostosis affecting the left arm is reported. This is the twenty-sixth case reported in the medical literature. The author believes that a previous skull injury with trauma to a nerve trunk was responsible for the melorheostosis. He believes that a parathyroid dysfunction was caused by the skull trauma and that the melorheostosis developed secondarily to this dysfunction. Emphasis is laid on the importance of trauma to the skull in melorheostosis.—*Mary Frances Vastine, M.D.*

Alderson, B. Roxby. Further observations on fracture of the first rib. *Brit. J. Radiol.*, Sept., 1947, *20*, 345–359.

Many variations in the first rib have been shown in mass chest examinations and these have been described as fracture, anomaly, synchrondrosis, pseudarthrosis, anomalous development simulating fracture, and idiopathic fracture. Actually fracture of the first rib is much more common than is generally accepted. It occurs frequently from muscular violence, and may result in complete union, or nonunion and formation of a false joint. Often there is sufficient pain for a patient to consult a physician, but more frequently there is not. A series of 77,607 British males was examined and 73

fractures of the first rib were found. Four times as many were seen in individuals between the ages of seventeen and twenty as in those over twenty-five years. If the abnormalities described were congenital or developmental the incidence should be the same in the two groups. Approximately 70 per cent of the cases were available for interrogation. Of these one-quarter admitted symptoms referable to the area. Of the rest, with no history, one-third had previous or subsequent roentgenograms which, when submitted to comparison, revealed either a previously normal rib or evidence of subsequent healing.

These fractures are rarely the result of pressure transmitted from the clavicle. The great majority are best classified as stress fractures and are due to muscle strain.—*E. F. Lang, M.D.*

BRAILSFORD, JAMES F. Some experiences with bone tumours. *Brit. J. Radiol.*, April, 1947, *20*, 129–144.

In the diagnosis of bone tumors a combination of clinical and roentgenographic evidence alone permits of the most accurate diagnosis and this evidence can be obtained without causing the patient pain and without disturbing the lesion, as by biopsy. In some cases repeated examination and observation are necessary before a definite decision can be made, but this is not a serious disadvantage and may even be an advantage. In the author's experience when the clinical or roentgenographic evidence is indefinite the histopathological evidence is at least equally indefinite and may only add confusion. The only time a biopsy is excusable is when it provides material to support clinical and roentgenological evidence of simplicity. On the other hand, amputation on the basis of a biopsy which shows malignancy without conclusive other evidence is to take an unjustifiable risk. Experience with cases which underwent amputation for malignancy and with those in whom amputation for a lesion which was subsequently found to be benign indicates that there is nothing to be gained by undue haste and precipitate amputation, but much can be gained by observation of the clinical features and serial roentgenograms. The author has not seen a cure result from amputation when there is typical roentgen evidence of an osteogenic sarcoma.

There is also the question of who is to interpret the films. The growing tendency for the clinician to use his own interpretation is to be decried. The radiologist, by learning and practice, has fitted himself for this. The clinician can make the roentgen examination most valuable by supplying important information which the radiologist must consider in his study of the films if he is to interpret correctly the roentgen changes in each case. In an attempt to develop young radiologists of the type who can best aid the clinician in his problems it is essential that these young men be given a thorough knowledge of all the roentgen features met with in all the specialties. The author believes this training should be given at the expense of the unnecessary training in radiation physics.

The principles stated above in the management of bone tumors are illustrated by a presentation of cases with excellent reproductions of their roentgenograms, and a critical discussion of each csae.

A fourteen year old girl who had suffered a blow to her arm seven weeks previously and whose roentgenogram had been interpreted elsewhere as showing Ewing's sarcoma was seen and conservative therapy was advised. The lesion disappeared on antiluetic therapy. The basis for conservative treatment was the impression that the appearance of the bone seven weeks after trauma was not consistent with the effects of trauma for that period. The time factor is important in determining whether or not a given bone lesion could be due to antecedent trauma.

A six year old girl was seen with a painful swelling over the upper part of the right arm with disintegration of the structure of the humerus, periosteal reaction, and multiple rounded secondary pulmonary lesions. Since surgery was hopeless, sulfathiazole was advised, but without apparent benefit as shown on serial film studies. Three months later, however, improvement began, and three months after this an examination showed resolution of the osseous changes and complete disappearance of the pulmonary lesions.

A twenty-five year old male was seen complaining of a lump in the midthigh which was not tender but which was associated with some destruction of the femur and new bone formation, suggesting malignancy. Roentgen therapy was administered. One year later a somewhat similar lesion was observed in the upper portion of the femur and this again was treated by irradiation. Six months later a destructive lesion developed in the left transverse process of the

fifth lumbar vertebra. The limb was amputated; the section showed secondary carcinoma, probably of a bronchial origin. Six months later another lumbar transverse process was involved. Six months later there were multiple metastases in the ribs, vertebrae, and pelvis. Chest films had been made since the patient had first been seen but it was not until this time, two years and nine months after the first metastasis, that lesions were found in the pleura from the primary in the lung.

Other instructive cases indicating the value of careful consideration and correlation of the clinical findings and serial roentgen examinations, which cases illustrate the wisdom of the avoidance of biopsy or other disturbance of the bone lesion, are included.—*E. F. Lang, M.D.*

Shackman, Ralph, and Harrison, C. V. Occult bone metastases. *Brit. J. Surg.*, April, 1948, *35*, 385–389.

In over half of the patients dead of malignant disease with bone metastases which were examined at autopsy, gross evidence of metastatic tumor tissue in bone was present, although roentgenographic examination before death and direct contact radiographs made of the bone before section revealed normal bony architecture. Four cases are presented with microphotographs, contact radiographs, and colored photographs of the sectioned bone. Blood borne metastatic growths are situated initially in the bone marrow, most frequently in bones containing red marrow. True bone change which is secondary to the presence of cancer cells is effected by the osteoblasts and osteoclasts and occurs only after the marrow spaces have been infiltrated and probably filled. Roentgenograms taken during life at greater distances and through superimposed soft tissues would be less likely to reveal metastatic deposits. So-called normal roentgenograms cannot exclude the presence of even extensive bone metastases.—*Charles W. Werley, M.D.*

Hitchcock, Harold H., and Bechtol, Charles O. Painful shoulder; observations on the role of the tendon of the long head of the biceps brachii in its causation. *J. Bone & Joint Surg.*, April, 1948, *30A*, 263–273.

The authors state that lesions affecting the tendon of the long head of the biceps brachii are among the more frequent causes of pain and disability in the region of the shoulder joint. In the majority of cases, common predetermining factors are responsible for the several pathological conditions encountered. Of these factors, the most significant are the presence of the "supratubercular ridge" of Meyer, described in 1928 and a preternaturally shallow bicipital or intertubercular sulcus which, with excessive function or as the outcome of trauma, results in a variety of lesions. These include acute or chronic peritendinitis, varying degrees of attrition and damage to the tendon, and its partial or complete dislocation.

The supratubercular ridge of Meyer is a ridge of bone, projecting immediately proximal to the medial wall of the bicipital groove and continuous with it. The biceps tendon is limited by this structure from slipping inward and is forced upward against the transverse humeral ligament, thus favoring dislocation. In their study, the authors included all ridges which they judged were high enough to force the biceps tendon against the roof of the groove and thus cause trauma to the tendon. They conclude that the normal anatomical mechanics of the biceps tendon are faulty. A supratubercular ridge or flat bicipital groove increases the mechanical difficulties.

An operation to fix the tendon in the bicipital groove and to remove that portion of the tendon above the groove relieves the symptoms caused by it and does not materially weaken the shoulder.—*R. S. Bromer, M. D.*

Davies, H. The unstable semilunar bone. *Lancet*, May 29, 1948, *1*, 831.

The carpal semilunar bone is not locked in its ball and socket position between the os magnum and the radius. It occupies a medial unstable position centered over the radioulnar joint. Its dorsal surface is reduced in size, and its articular facet for the os magnum lies obliquely. All these factors contribute to its instability and explain how pressure through the os magnum can so readily produce dislocation. It is remarkable that medial dislocation does not occur more often.—*J. S. Summers, M.D.*

Immermann, E. William. Dislocation of the pisiform. *J. Bone & Joint Surg.*, April, 1948, *30A*, 489–492.

Dislocation of the pisiform is a rare entity. It is still more rare to have chronic recurrent dislocation of the pisiform. The author believes

his case to be the first complete description of such an entity.

Dislocation of the pisiform may result either from a direct injury or from muscular violence. In the cases reported in the literature, the ratio was two to one in favor of muscular force as an etiological agent. This force in all instances was manifested as the patient was in the process of lifting a heavy object. Clinically, following a dislocation, the pisiform is painfully displaced from its normal site, leaving a depression. Flexion and adduction may be painful, and there may be exaggerated mobility of the bone. The diagnosis can be confirmed by roentgenograms, with the ulnar side of the wrist placed against the film in about 15 degrees of supinated rotation. It must be borne in mind, however, that in an acute or recurrent dislocation the bone may return to its normal position, producing a negative roentgenogram. In the chronic case, such as that of the author, where the patient complains of pain in the volar aspect, of the wrist with a sensation of "giving away" upon lifting, duplication of the etiology may confirm the supposed diagnosis.

In acute cases which do not respond to splinting, and in chronic cases, surgical excision is indicated. Removal of the bone in no way interferes with the normal function of the wrist. In an acute dislocation, the bone should be manipulated into place, and splinting should be applied using an anterior molded plaster splint.—*R. S. Bromer, M.D.*

ANDREASEN, A. T. Congenital absence of the humeral head; report of two cases. *J. Bone & Joint Surg.*, May, 1948, *30B*, 333–337.

The author states that the purpose of this article is to present clinical data, review the literature, and speculate as to the origin of a rare condition. The first case, he reports, had been passed over with the remark that "the condition is probably congenital." Attention was aroused when a second case was seen a few months later. Only 6 cases with similar roentgen appearances could be collected from the literature. In most of the cases other skeletal deformities were present, whereas in his cases only the shoulder was affected. The essential feature of the 2 cases was complete or incomplete absence of the humeral head with a rudimentary glenoid. The deformity may be unilateral or bilateral. It may be isolated or it may be one of a number of deformities in the same patient. It is associated with relatively slight disability. The ball and cup arrangement of the shoulder joint is reversed or it is replaced by a saddle-shaped articulation. There seems to be correspondence between the degree of capital deficiency and the state of development of the glenoid. It appears that lack of the capital epiphysis, or its cartilaginous and precartilaginous stages, causes a corresponding lack of the upper epiphysis of the glenoid-complex while the main body of the glenoid continues to develop.

Consideration of his 2 cases, together with experimental data from the studies of Fell and Canti, suggests that the time in development at which the fault occurred was the presumptive joint stage, just when the articular rudiments had separated.—*R. S. Bromer, M. D.*

MCDONNELL, D. P., and WILSON, JOHN C. Fractures of the lower end of the humerus in children. *J. Bone & Joint Surg.*, April, 1948, *30A*, 347–358.

This paper is based on a study of 176 fractures of the lower end of the humerus in children. Eighty-eight of the number were followed long enough to draw some general conclusions with regard to treatment and complications. Of the latter group, 55 were in boys and 33 in girls. The ages of the patients ranged from one to twelve years, the average age being seven. There were 53 supracondylar fractures, 33 lateral condylar fractures, 1 medial epicondylar fracture, and 1 epiphyseal separation of the capitellum.

Contrary to prevailing opinion, the authors believe supracondylar fractures can produce serious growth disturbance if the fracture lines extend into the epiphyseal centers. Cubitus varus and cubitus valgus are not considered serious disabilities, if flexion and extension are complete. However, if the deformity is progressive and is caused by disturbances in the growth centers, disabilities and limitation of motion may occur years after the initial injury. Supracondylar fractures in which the fracture line, or part of it, extends into the epiphyses may produce growth disturbances similar to those which result from fractures of the lateral and medial condyles. It the fragment is intra-articular and not attached to the overlying bone and soft tissue, it will undergo vascular necrosis. Fractures of this type should have an accurate anatomical reduction, and if the fragments are displaced and rotated, an open re-

duction is probably the best method of obtaining accurate alignment.

In lateral condylar fractures, avascular necrosis of the capitellum will occur if the fracture line separates the epiphysis from the overlying bone and soft tissues. Early open reduction with minimal trauma to soft tissues will give the best results. Fractures of half of the capitellum occurred in 2 patients with lateral condylar fractures. One had poor apposition by open reduction, and avascular necrosis occurred in one year. In the other, the separation was not complete, the position was good, and healing was uneventful.

Avascular necrosis of the trochlea may occur, later, despite good reduction of a supracondylar fracture. It can cause stiffness at the joint, due to thinning of the cartilage and distortion of the articular surface of the humerus.

Delayed ulnar palsy may cause progressive growth disturbances, and was present in 2 cases of severe cubitus valgus deformity.

Fracture of the medial epicondyle will not cause a serious disability unless the fragment enters the joint cavity. Non-union is difficult to avoid in closed reduction.—*R. S. Bromer, M.D.*

Andreasen, A. T. Avulsion fracture of lesser tuberosity of humerus; report of a case. *Lancet*, May 15, 1948, *1*, 750.

A woman fifty-six years of age was dismounting from a side saddle when her foot slipped. Since the right hand retained a grip on the saddle post, there was trauma to the shoulder with the arm fully abducted above the shoulder, the hand being fixed, and the body moving. This resulted in an avulsion fracture of the lesser tuberosity as proved by roengtenograms.

Operation was refused by the patient. The bone fragment first underwent aseptic necrosis, then was adsorbed in fourteen months. A study of the literature failed to reveal any similar case and this is thought to be a rare injury. Reproductions of the roentgenograms clearly show the avulsed fragment lying inferoposteriorly to the head of the humerus.—*J. S. Summers, M. D.*

Shellito, John G., and Dockerty, Malcolm B. Cartilaginous tumors of the hand. *Surg., Gynec. & Obst.*, April, 1948, *86*, 465–472.

Cartilaginous tumors of the hand (1) interfere with function, (2) are a source of potential malignancy, and (3) are unsightly for cosmetic reasons. It was found, on reviewing the literature, that all too often cartilaginous tumors are lumped together under the heading "osteochondroma" and the suffix "sarcoma," added to denote malignancy. It was discovered that it is correct, on the basis of pathology, to divide these tumors into two types: the enchondroma, or chondroma, and the ecchondroma.

Roentgenographic Observations

Enchondroma. The roentgenographic picture of these tumors caused the roentgenologist to suspect the presence of chondroma in 50 per cent of the cases. The typical picture was that of the so-called bone cyst which was seen as a small, round area of decreased density in the center of the bone. The cortex usually was expanded and thinner than it normally is. The outline was round and regular and appeared in the center of the shaft.

Ecchondroma. In contrast, the roentgenologist was able to make the diagnosis in almost all of the cases of ecchondroma. The lesion in this case appears at the periphery and one end of the bone. The bony elements of the base of the ecchondroma show plainly as an elevation of bony structure.

Summary and Conclusions

1. Cartilaginous tumors of the hand, on the basis of pathology, may be divided into two types: enchondroma and ecchondroma. The synonym for enchondroma is chondroma. Ecchondroma is called "perichondroma," "osteochondroma," and "exostosis." (The word "osteochondroma" is restricted to the ecchondroma.)

2. The fact that the entire group of cartilaginous tumors is commonly called osteochondroma has led, both in the past and in the present, to a great deal of confusion. It is better to use the terms enchondroma and ecchondroma.

3. Distinction between the two cannot be made with slide and microscope alone.

4. An enchondroma is usually a small cartilaginous tumor located centrally in the bone shaft, growing from many centers and expanding in all directions. Rate of incidence of pathologic fracture in our series was 26.6 per cent. The enchondroma occurs much more often in the hand than does the ecchondroma (3:1). These tumors, in comparison to normal hyaline

cartilage, show increased vascularity, calcium deposition, and myxomatous degeneration.

5. Two tumors of the tendon sheaths were found, both of which were enchondromas. In one of these cases multiple enchondromas of the sheath had developed and this is good evidence that an enchondroma can occur in the tendon sheath away from the site of tendon insertion.

6. The ecchondromas occur peripherally in bone near the end of the shaft and have a bony base and cartilaginous cap.

7. Either type of tumor may be malignant, in which case it is usually a sarcoma. In this series 2 per cent were malignant. Both types are prone to recur, although recurrence does not mean malignancy unless a mitotic figure can be found.

8. The treatment in both types is conservative operation. —*Mary Frances Vastine, M.D.*

UMANSKY, A. L., SCHLESINGER, P. T., and GREENBERG, B. B. Tuberculous dactylitis in the adult. *Arch. Surg.*, Jan, 1947, *54*, 67–78.

Tuberculous dactylitis or spina ventosa is usually a disease of infancy and childhood and occurs uncommonly in adults.

The authors describe in detail their experiences in a nineteen year old Negro in whom a tuberculous dactylitis was followed over a period of approximately two years following which the finger was amputated and submitted for histopathologic study.

Then follows an excellent discussion of the etiology, pathogenesis, and roentgen characteristics of tuberculous dactylitis.

According to the authors, the earliest roentgen evidence of tuberculous bone involvement in spina ventosa is elevation of the periosteum. Usually the result of an underlying serous effusion, the elevation is manifested by a linear deposit of new bone surrounding part or all of the diaphysis. Soon thereafter cortical changes may be demonstrated by an indefinite bone irregularity. Although at first swelling of the soft tissues is unusual, this soon occurs and progresses as the disease advances.

In children, the periosteal reaction is intense. The cortical expansion may be so distinct that the normal shaft may occasionally be seen within the expanded periosteal sheath of new bone. Gradually, however, the shaft is destroyed and separates as a sequestrum. The sequestrum then shows progressive fragmentation and absorption leaving a cyst-like cavity commonly called the spina ventosa. Often, however, the sequestrum may separate with necrosis of the new periosteum and fistula formation.

In adults, the initial periostitis is often replaced by a large area of rarefaction. The compact cortex becomes absorbed and the fine cancellous bone structure is replaced by much coarser cancellous patterns which often show some expansion. The bone cortex itself may be partially eroded or destroyed, with resultant sclerosis of the trabeculae in the adjacent portion of the shaft in which the cancellous bone tends to become obliterated. As the disease progresses, the cortical irregularity and sclerosis become more marked while its cancellous portion, already coarse, may develop irregular areas of rarefaction which finally appears honeycombed.

At this stage, pathologic fractures commonly develop. Finally, bone destruction may become so extensive that the debris may be seen in the perifocal soft tissues. The remaining portion of the shaft may become wide and sclerotic and the medullary cavity almost completely obliterated without the appearance of a large sequestrum, involucrum, or fistula.

The greater incidence of tuberculous dactylitis in childhood is attributed to the vascularity of these short tubular bones in the first four or five years of life. Also important is the marked regenerative power of the periosteum which is far less active in adult life.

The reproductions of the roentgenograms of the affected finger are excellent and clearly depict the progressive character of the tuberculous dactylitis in an adult from its earliest stages when only periosteal reaction was evident until marked bone destruction with a pathological fracture forced its amputation.

A fairly complete bibliography adds to the value of this review.—*Philip J. Hodes, M.D.*

BERK, MORTON E. Gout; report of an unusual case in a young man. *Am. J. M. Sc.*, March, 1948, *215*, 290–295.

Report of a case of a twenty-eight year old Filipino with proved gouty arthritis. History of onset prior to his eleventh birthday. The patient's diet was never high in purines and hyperuricemia was not present in several laboratory determinations. A tophus was found behind the ear.—*Russell R. Jauernig, M.D.*

Burrows, H. Jackson. Fatigue fractures of the fibula. *J. Bone & Joint Surg.*, May, 1948, *30B*, 266–279.

The paper consists of a review of fatigue fractures of the fibula and the author adds 5 more. The clinical and roentgen features, diagnosis, treatment and results are discussed and special note is made of the misleading freedom of ankle and tarsal movements and the occasional absence of tenderness. The fractures in the lower third occur particularly in two groups of subjects: (1) young male runners and skaters; (2) active and hard-pressed women of middle age and over. In male runners and skaters the fracture usually occurs through slender, mainly cancellous bone, 2 inches or more above the tip of the lateral malleolus; in middle-aged women the fracture is usually distal to the interosseous ligament through thicker, mainly cancellous bone, 1½ inches from the tip of the lateral malleolus. The most convenient name, Burrows suggests, is low fatigue fracture of the fibula.

His review of the literature of fatigue fracture of the uppermost third of the fibula shows that it is often precipitated by jumping. The most convenient name for it is high fatigue fracture of the fibula. Like all clinical classifications this distinction between low and high fractures has exceptions. Both high and low fractures may be bilateral.

During the first fifteen days or more no roentgen changes may be discernible. On the other hand, a fracture line may be evident after a week; sometimes none appears till the end of the second or third week. At first it is difficult to see, and even after four weeks it may be recognized only with the aid of a lens. A band of rarefaction may be seen at twelve to eighteen weeks. Callus has been observed in a child as early as the eighth day. In adults it appears during the third week, steadily consolidating and becoming organized at twelve to sixteen weeks when it forms a spindle-like thickening, crossed by a less dense stripe at the site of fracture. At forty-seven weeks it may still be just possible to distinguish thickening. A dense band is often the first indication of the fracture site and almost the last to disappear. The plane of fracture is almost horizontal; slight deviations from this are not always in the same direction so that little can be concluded about the fracturing stresses.—*R. S. Bromer, M.D.*

Rolleston, G. L., The early radiological diagnosis of ankylosing spondylitis. *Brit. J. Radiol.*, July, 1947, *20*, 288–293.

The earlier ankylosing spondylitis is recognized, the more amenable it is to treatment. The earliest appearance of the disease occurs in the sacroiliac joints, and later changes are seen at the margins of the vertebral bodies and in the costotransverse articulations.

Roentgen examinations of patients with symptoms suggesting lumbar vertebral or sacroiliac origin may be divided into four groups:

1. Anatomical abnormalities.
2. Definite pathological entities of bones or joints.
3. The frankly negative for any recognizable cause for symptoms; this group is by far the largest.
4. The borderline departures from normality. It is in this last group that the earliest changes of ankylosing spondylitis are found.

In the sacroiliac joints there is first seen loss of the normal definition of the subarticular cortex in patches. The joint margins in areas fade off into the rest of the bone. This is due to small subarticular erosions. In the superior vertebral angle of the ilium there is loss of the superficial cortical layer, producing a ragged, ill-defined edge. As the changes progress the joint margins become more irregular with a moth-eaten appearance and apparent widening of the joint space. The disease also involved the superior horizontal portion of the sacrum. As the disease progresses, there is progressive loss of the joint space, and temporary mottled calcium deposits are seen in the juxta-articular ilium and sacrum. The final stage is complete ankylosis with absorption of the sclerotic bone and relatively normal appearing cancellous bone.

In the vertebral bodies the changes may progress either up or down, and there may be involvement of the cervical and lumbar regions with initial sparing of intervening vertebrae. The earliest changes are seen in the posterior intervertebral joints, but special views are necessary to see these. In the routine films the first sign is loss of the normal convex anterior contour with sharpening of the upper and lower anterior margins. Later there is filling-in of the anterior concavity and the final stage is the development of the "bamboo spine."

The upper costotransverse joints are fre-

quently well seen in posteroanterior chest films and anteroposterior views of the cervical spine. The articular surfaces become irregular with erosions and osteoporosis. In some cases this may limit chest expansion and be one of the first signs of the disease.—*E. F. Lang, M.D.*

BARNES, ROLAND. Paraplegia in cervical spine injuries. *J. Bone & Joint Surg.*, May, 1948, *30B*, 234–244.

Twenty-two cases of paraplegia complicating injury of the cervical column are reviewed in this paper. The vertebral injury is due to flexion or hyperextension violence. Of the flexion injury, Barnes recognizes three types: (1) dislocation; (2) compression fracture of a vertebral body; (3) acute retropulsion of an intervertebral disc. He presents evidence to support the view that disc protrusion is the cause of the cord lesion when there is no roentgen evidence of bone injury, and in some cases at least when there is a compression fracture.

Two types of hyperextension injury are described: (1) dislocation; (1) injury to arthritic spines. Hyperextension injury of an arthritic spine, he considers, is the usual cause of paraplegia in patients over fifty years of age.

In one of the cases of flexion injury, the roentgenographic examination showed no evidence of bone injury. The intervertebral disc between the third and fourth cervical vertebrae was narrowed. Myelography after injection of lipiodol into the lumbar area showed complete block at the level of the herniated disc. The patient, who was examined an hour after the accident, had incomplete paralysis and sensory loss in the lower limbs, trunk and upper limbs.

Treatment of flexion injuries is discussed and Barnes presents the indications for caliper traction and laminectomy.

Elderly patients are predisposed to hyperextension injury by two factors. With advancing years there is a tendency to kyphosis of the thoracic spine which is usually compensated by a cervical lordosis, an extension deformity of the neck. This is a relatively fixed position and any increase in the degree of extension is likely to cause strain or injury. The risk of injury by hyperextension violence is greater when mobility of the spine is impaired by arthritic changes in joints and ligaments. In the event of a fall, the neck cannot be flexed in the normal defensive manner, so that when the head strikes the ground powerful angulation strain in extension is applied to the cervical region. The brunt of this violence may fall on the anterior common ligament and it may rupture, or a small flake of bone may be avulsed from the lower anterior margin of the upper vertebra. The tear extends backwards through the intervertebral disc, or the disc itself may be torn from the vertebra above. There is no tendency for the disc to herniate into the spinal canal as in flexion injuries. Once the disc has ruptured there is little resistance to further hyperextension, and considerable separation of the vertebral bodies can occur without rupture of the lax posterior common ligament or the zygapophyseal joint capsules. Immediately the striking force is removed normal alignment of the vertebrae is restored by spasm of the neck muscles and for this reason the roentgen appearances may be deceptive.

Barnes has been unable to find a completely satisfactory explanation of a spinal cord lesion in these hyperextension injuries. He suggests two possible explanations: (1) that it may be caused by "spinal concussion" as the result of minor displacement of a vertebral body; or (2) that it may be due to traction injury at the moment of extreme hyperextension.

Caliper traction is regarded as unnecessary in hyperextension injury of an arthritic cervical spine. The intervertebral disc is not displaced backward into the spinal canal and any vetebral displacement that may be present is easily corrected by moderate flexion of the neck. External splintage is not necessary and elderly patients are so intolerant of plaster jackets or collars that their application often hastens the fatal termination.—*R. S. Bromer, M.D.*

TAYLOR, ALEXANDER R., and BLACKWOOD, WILLIAM. Paraplegia in hyperextension cervical injuries with normal radiographic appearances. *J. Bone & Joint Surg.*, May, 1948. *30B*, 245–248.

A case is reported of paraplegia with normal roentgen appearances in which the cervical cord damage at autopsy was shown to have been due to hyperextension injury. In the view of the authors, the diagnosis of such cases lies between extension dislocation with immediate spontaneous reduction, and acute prolapse of an intervertebral disc. The history may clearly indicate whether the injury took place in flexion or extension. Older patients with fixed kyphotic

deformity are liable to extension injury. Disc protrusion may or may not cause spinal block, and although hyperextension dislocation does not result in continued narrowing of the spinal canal at the point of injury, yet spinal block may develop later by reason of edema of the cord due to contusion.

Roentgen examination may assist in the diagnosis by disclosing minor changes. Narrowing of a disc space, or the suspicion of wedging of a vertebra, creates a strong presumption that the compressing agent is a ruptured disc. The tearing of a small flake of bone from the anterior margin of a vertebral body suggests rupture of the anterior longitudinal ligament, and therefore extension injury. If doubt exists, myeography should dispel it, because a prolapsed disc of sufficient size to damage the cord should be visualized without difficulty.

The authors suggest that hyperextension is not the only one of the mechanisms of injury in this type of case but that it is the usual mechanism. They state that the practical value of the recognition of hyperextension injuries lies in treatment. The extended position in which, by well established usage, all cases of traumatic cervical paraplegia are treated is in fact the position most calculated to inflict further damage to the injured cord. They suggest that if the diagnosis of hyperextension injury is made, the patient should be nursed in a shell, or between sandbags, in the neutral position or even in a position of slight flexion.—*R. S. Bromer, M.D.*

Kristoff, Frederic V., and Odom, Guy L. Ruptured intervertebral disk in the cervical region. *Arch. Surg.*, March, 1947, *54*, 287–304.

The authors report 20 cases of ruptured intervertebral cervical disk. Their experiences have led them to believe that there are three successive stages in the pathogenesis of ruptured cervical disks. The first period is that of root compression. The second period is that of unilateral cord compression with or without masked root compression. The final or third period is that of bilateral cord compression with masked unilateral or bilateral root compression.

The authors believe the diagnosis of cervical disc lesions is primarily clinical and that the history and subjective complaints are of the greatest assistance in early diagnosis. Considerable emphasis is placed upon the diagnostic significance of being able to reproduce the pain and paresthesias complained of by the patient by manipulation of the lower cervical spine.

In the cervical canal, there is a much closer relationship between the cervical interspaces and their corresponding nerve roots. Because of this, root compression in the cervical canal is localized with much greater accuracy than in the lumbar spine. Of importance, too, is the fact that in the cervical canal, the spinal cord is anchored on both sides by the dentate ligament which tends to fix the cervical neural structures thereby accounting for the rapidity of onset of signs and symptoms in relatively small protrusions of the disk.

Ruptured disks at the 5th cervical interspace produce pain and paresthesias in the radial side of the arm, thumb and index finger. Sensory impairment, if present, may involve the same area. There may be associated weakness of the deltoid and biceps muscles.

Ruptures at the 6th cervical interspace are attended by pain and paresthesias referable to the extensor side of the arm, dorsum of the hand and one or all of the three middle fingers. Sensory impairment, if present, may involve the same area or part thereof. Weakness primarily involves the triceps muscle.

Roentgenograms of the cervical spine are of questionable value in these lesions. In some instances, the physiological cervical lordotic curve may be absent. Oblique views of the cervical spine may or may not show localized arthritic changes and narrowing of the intervertebral foramina.

The authors re-emphasize the fact that apparently normal roentgenograms of the cervical spine are not uncommon in patients with proved ruptured cervical intervertebral disks.

The importance of myelography and the accuracy of the method is re-emphasized.

The authors divide their discussion into two sections depending upon whether the lateral intervertebral disk protrusions with root compression are complicated by or are uncomplicated by cord compression.—*Philip J. Hodes, M.D.*

Peyton, William T., and Simmons, Donald R. Herniated intervertebral disk. *Arch. Surg.*, Sept., 1947, *55*, 271–287.

The authors report their experiences in 90 patients operated upon for herniated interver-

tebral disks. In 10, the intervertebral disk was considered normal at operation. In 80 patients, the intervertebral disk was considered abnormal. To be considered abnormal or pathologic, a portion of the disk had to lie loose as a fragment of tissue in the spinal canal or had to protrude spontaneously when the annulus fibrosus was opened.

The results were considered satisfactory in most of the patients in whom a definitely pathologic disk was found and removed. Seventy-two of the 80 patients in this latter group were followed and in only 3 was continued pain or disability reported. The results were uniformly unsatisfactory in the 10 patients in whom a normal intervertebral disk was found.

During the authors' early experience spinal fusions were not performed after the disk was removed. More recently, however, routine spinal fusions accompanied every disk operation. The results indicated that when truly pathologic intervertebral disks were operated upon, it made little difference whether or not the removal of the intervertebral disk was accompanied by spinal fusion.

Routine roentgenograms of the lumbosacral portion of the spine seemed to indicate that bony abnormalities were not predisposing causes of herniation of intervertebral disks in the present series. Of the 90 patients examined roentgenographically, in 16 narrowing of the interspace involved by the pathologic disks was reported. Of equal significance, however, was the occurrence of similar narrowing at levels other than the one involved by the pathologic disk.

Discussing contrast studies of the spinal canal, the authors report that they discarded air myelography in 1941 because they thought the procedure was unreliable. Since then, they have used radiopaque substances (pantopaque) in selected cases only. Many patients with classical symptoms were not examined in this manner since operation would have been performed even though the myelograms proved to be negative. In other patients in whom the findings were less definite, the contrast study of the spinal canal was carried out in the hope of obtaining additional evidence.

Contrast myelograms were obtained in 46 of the 90 patients operated upon. A correct diagnosis was made in 41 instances. In 3, the roentgen diagnosis was wrong.—*Philip J. Hodes, M.D.*

CLEVELAND, MATHER, BOSWORTH, DAVID M., and THOMPSON, FREDERICK R. Pseudarthrosis in the lumbosacral spine. *J. Bone & Joint Surg.*, April 1948, *30A*, 302–312.

The paper is divided into two parts: first, the incidence of pseudarthrosis in fusion of the lumbosacral spine; and second, statistics regarding the repair of pseudarthrosis. Among 594 patients, 1,329 spinal intervals were bridged. Pseudarthrosis developed in 161 intervals among these patients, or 12.1 per cent. Among the 594 patients, 119 developed pseudarthrosis, or 20 per cent. It was found that the percentage of pseudarthrosis rose rapidly as the number of spinal intervals, which were crossed, increased.

Failure of fusion was demonstrated much more frequently when roentgenograms of front and side bending (biplane) were superimposed than by use of flat roentgenograms and clinical observation. In these biplane roentgenograms, the lateral flexion and extension films should show the fused elements completely superimposed. In the superimposed anteroposterior lateral-bending roentgenograms, the fused elements appear as if stereoscoped, but the borders of the bodies of the spinous and transverse processes are parallel. The use of such superimposed roentgenograms has increased the known percentage of pseudarthrosis, when tabulated both as to clinical incidence and true incidence. In the study of the authors' cases no result was accepted as regards solidity of fusion with less than one year of follow-up, unless controlled by both lateral roentgenograms in flexion and extension and by anteroposterior roentgenograms in right and left bending.

Of the 119 patients in whom pseudarthrosis developed, only 35 returned to the authors for repair. Many of the remainder had symptoms sufficient for repair. These cases are analyzed in detail in the paper. Relation of the failure of pseudarthrosis repair to the type of operation and causes for the development pseudarthrosis are also discussed.—*R. S. Bromer, M.D.*

HORWITZ, THOMAS. The treatment of congenital (or developmental) coxa vara. *Surg., Gynec. & Obst.*, July, 1948, *87*, 71–75.

This investigation is based on 17 cases of congenital or developmental coxa vara. This deformity should be suspected in any child who manifests a painless limp. Pain which appears in adolescence and in the adult is depend-

ent on the degree of deformity and shortening and upon the extent of degenerative changes which affect the hip joint (largely on a static basis). The roentgenograms and histologic studies in these cases show a pronounced disorganization of the femoral neck in the region of, and distal to, the epiphyseal plate. The exact nature of these changes is not clearly understood.

An evaluation of the results of surgery in these 17 cases of coxa vara fails to justify the use of drilling of, or introduction of a bone graft through, the femoral neck as a procedure supplemental to corrective osteotomy. The results have been satisfactory more consistently, with a lesser incidence of immediate postoperative complications, following simple subtrochanteric osteotomy than by any other method. Recurrence of coxa vara deformity after subtrochanteric or intertrochanteric osteotomy in 4 cases was due to mechanical difficulties. These difficulties involved the control of the postosteotomy fragments in the presence of a movable hip joint.—*Mary Frances Vastine, M.D.*

Bost, Frederic C., Hagey, Helen, Schottstaedt, Edwin R., and Larsen, Loren J. The results of treatment of congenital dislocation of the hip in infancy. *J. Bone & Joint Surg.*, April, 1948, *30A*, 454–468.

This paper is a study of the results of treatment of congenital dysplasia and of congenital dislocation of the hip in 112 such disorders which occurred in 86 patients, upon whom treatment was commenced in the first three years of life. The report is actually a study of the early results, covering a period of from one to thirteen years after the commencement of treatment. The types of disorders follow the suggestion of Hart, being designated as either dysplasia or as dislocation of the hip. The first term is applied to those hips which lack normal development of the roof of the acetabulum, and the second designates those hips in which dysplasia was found in conjunction with some outward or upward displacement of the head of the femur. No case of arthrogryposis was included in the series.

The authors regard roentgenography as the sole means by which the anatomical condition of the hip may be judged. The interpretation of the roentgenogram should include specific information concerning both the acetabulum and the head and neck of the femur, as well as their relation to one another. The measurements of Hilgenreiner were used as they satisfy these requirements. In this measurement the transverse or Y line extends between the tips of the ilia at the region of the triradiate cartilage. The slope of the acetabulum is measured from the tip of the ilium to the upper and outer portion of the acetabulum. The angle between the line marking the roof of the acetabulum and the Y line has been designated as the "acetabular index." A third line, drawn perpendicular from the Y line to the highest point of the upper femoral diaphysis, permits the measurement of the distance of the femoral diaphysis either upward or downward from the Y line, and also its distance outward from the tip of the ilium at the base of the acetabulum. The measurement of Perkins, which is a perpendicular line from the upper and outer border of the acetabulum to the Y line, is helpful in indicating the position of the head of the femur. The Y coordinate for the hip joint is the measurement from the line of gravity of the body, as represented by a longitudinal line through the mid-sacrum, to the center of the head of the femur.

If comparisons of roentgenograms are made, a standard technique must be used and the roentgen tube must be well centered over the pelvis which has been placed in a symmetrical position with the knees together and the patellae uppermost. When any doubt exists concerning the hip joint, the roentgenograms should be repeated at intervals. While the mathematical accuracy of the interpretation of the roentgenograms may be open to question, nevertheless the comparison of the two hips and of roentgenograms taken in series provides relative accuracy upon which judgment may be based.

Excellent or nearly excellent anatomical results were obtained in 55.4 per cent of the cases. Good anatomical results were produced by treatment in infancy in 81.3 per cent, and good functional results in 80.4 per cent of the hips examined. Development of the acetabulum progressed slowly, indicating a probable need for longer periods of fixation after reduction of the hip. Delay in reduction of dislocation was found to cause increasing maldevelopment of the hip, due in part to a thickening of the acetabular floor coincident with an increase in the Y coordinate. There was found, also, a direct relation between the early institution of treatment and the good results reported.—*R. S. Bromer, M.D.*

DONALDSON, S. W., BADGLEY, CARL E., and HUNSBERGER, W. G. Lateral view of the pelvis in examination for hip dislocation. *J. Bone & Joint Surg.*, April, 1948, *30A*, 512–514.

A technique is recommended for lateral view of the pelvis in examination for dislocation of the hip. Stereoscopic anteroposterior views of the pelvis and both hip joints do not always give complete information to determine the exact amount of displacement. The technique employed and the position of the patient for such a roentgenogram are the same as used for the lateral view in pelvic measurement examination, fractures of the sacrum and lesions of the lower lumbar spine and the lumbosacral disc. Routine roentgenograms of the pelvis and both hips are first made; and, if they show extensive fractures of the pelvis, then the lateral view is omitted for obvious reasons. If the roentgenograms reveal only a dislocation of the femoral head, or a dislocation with an acetabular fracture, the patient is placed in the proper position on the Bucky table for a lateral projection of the pelvis. Sometimes it is more comfortable for the patient to lie with the injured hip on the table top instead of away from it. The additional exposure not only shows the amount of displacement of the head of the femur, but also shows the position of any small bony fragments if present, either inside or outside of the joint capsule. Such a roentgenogram is also of value in the check-up examination following manipulation for reduction of a simple dislocation without fracture. The authors found that examinations with a portable apparatus, while the patient is in bed and extension applied, have not been satisfactory, either with or without a stationary grid.—*R. S. Bromer, M.D.*

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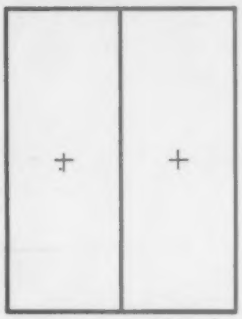

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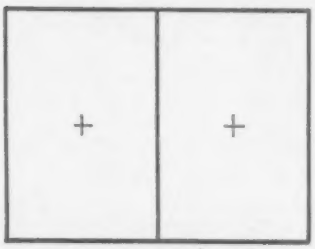

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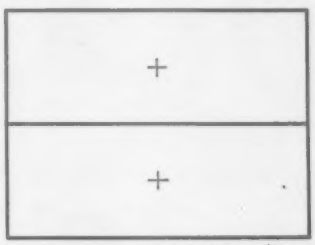

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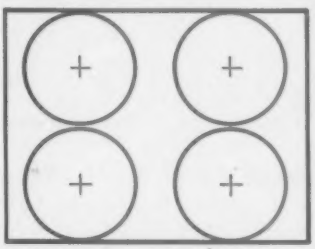

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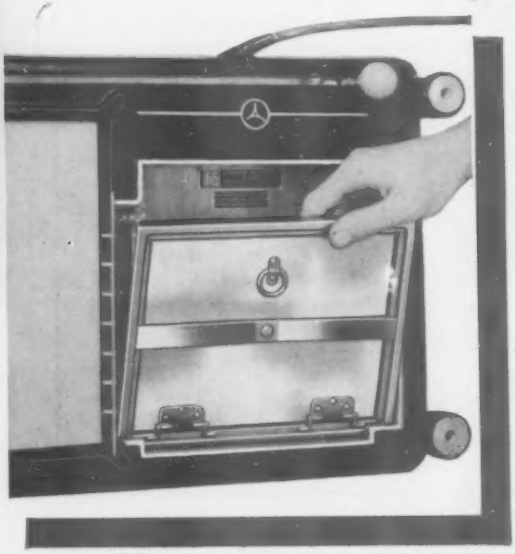

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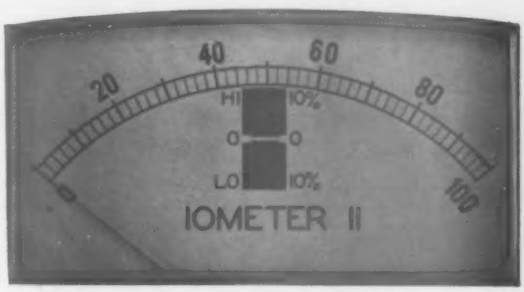

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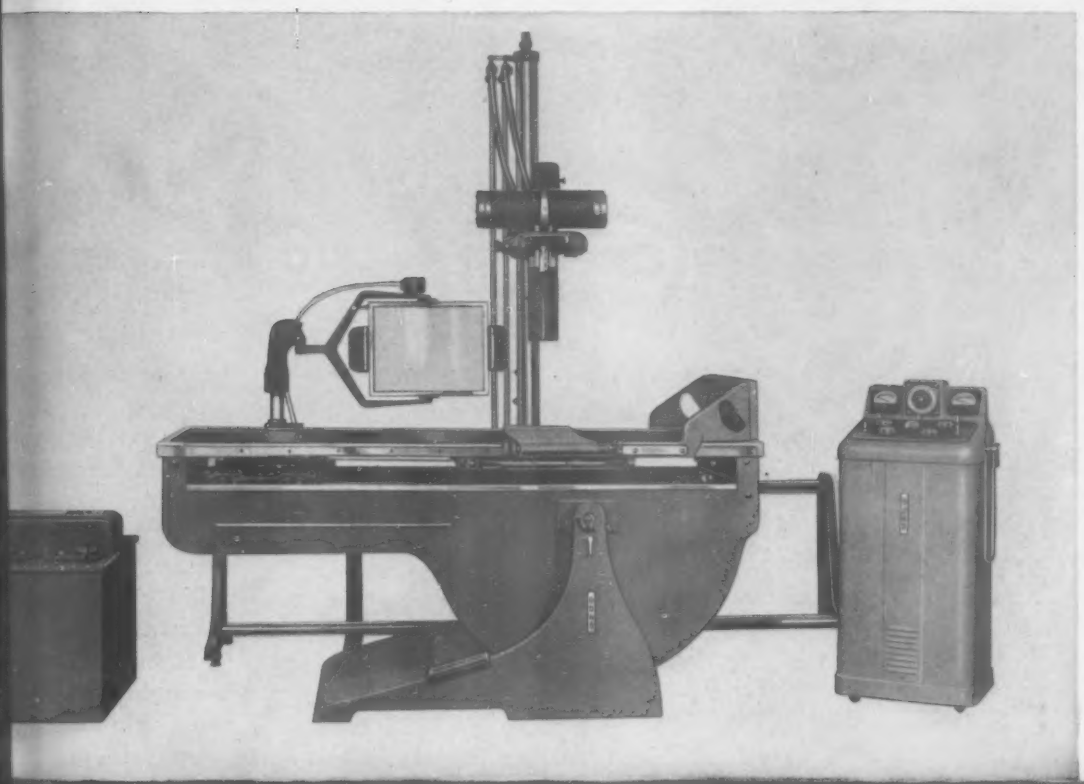

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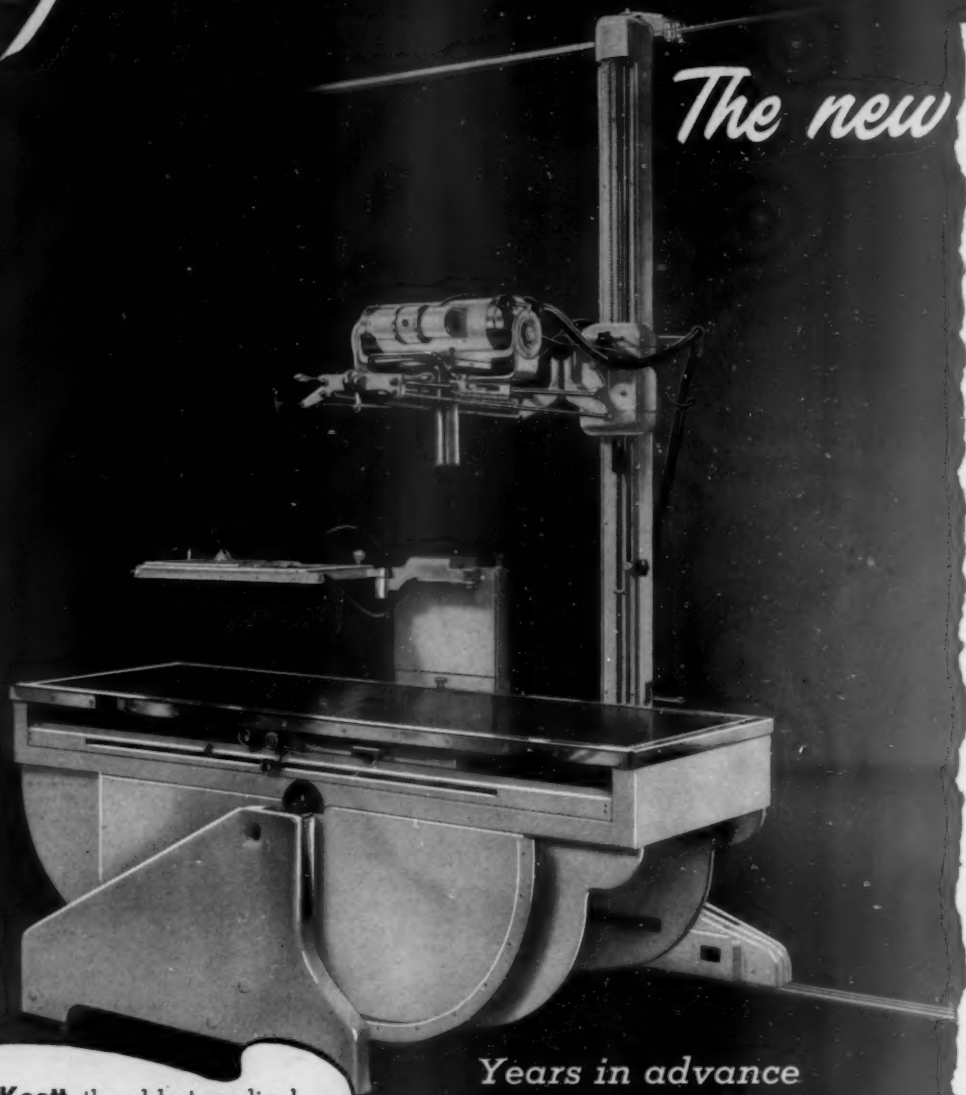

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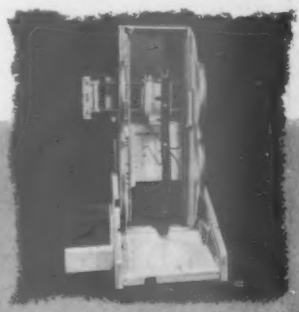

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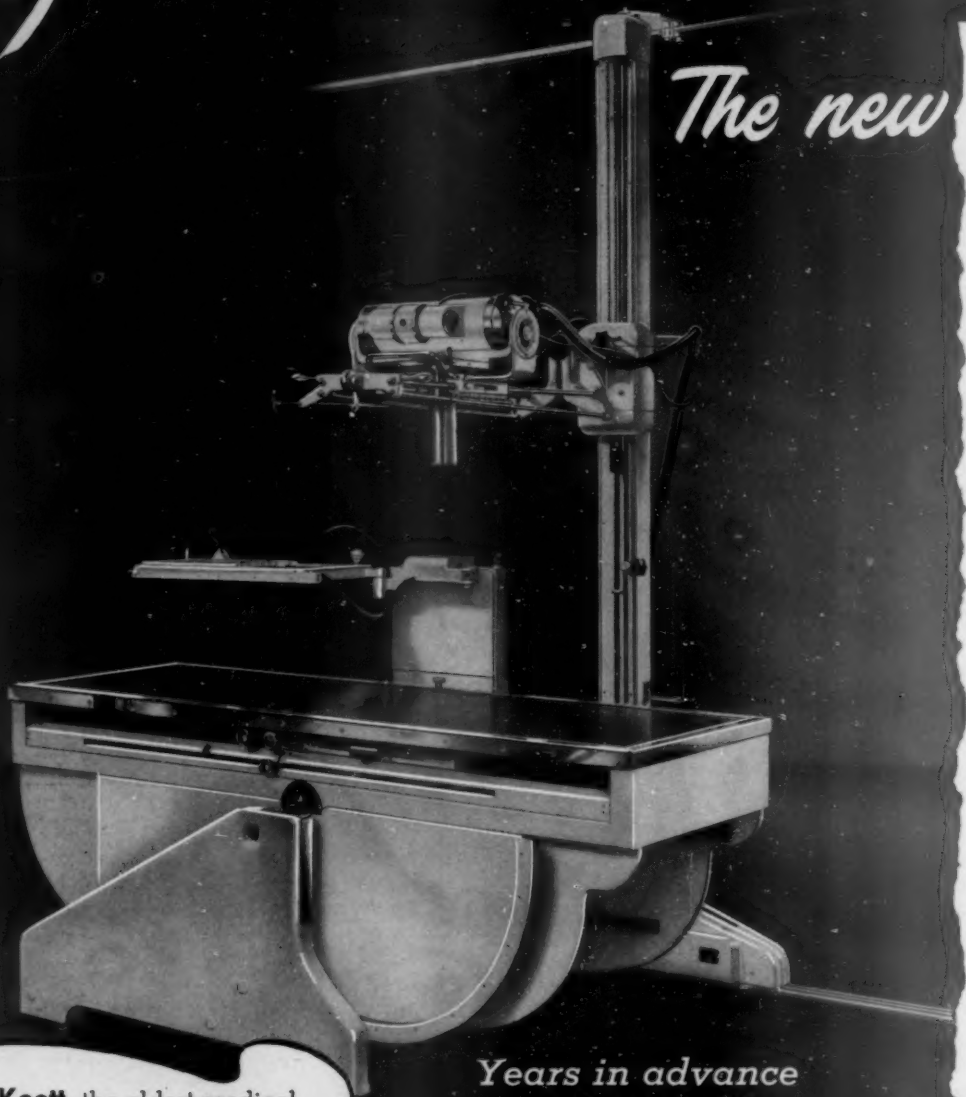

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11. Motor housing serves as observation platform.
12. Table is driven from both sides by double gear segment.
13. 135° of angulation . . . from 45° Trendelenburg through horizontal to vertical.
14. X-Ray absorption maximum of 0.5 MM of Aluminum at 60 PKV.
15. Table is impervious to water and diagnostic solutions.
16. No holes in table top . . . attachments are made on side of table.
17. High speed Bucky, 17″ x 17″, 50 line grid, 1/10 second to 30 second timing range.
18. Bucky covers up to 66″ of table range . . . to within 3″ of foot end.
19. Reciprocating Bucky available.
20. For Fluoroscopic work, uses Patterson B-2 screen and Scholz four and one spot film tunnel.
21. Fluoroscopic tower is disappearing type.
22. Fluoroscopic screen or spot film device has four positive locks for every technic.
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27. Cross travel of fluoroscopic tube is 10″, longitudinal—42″
28. Fluoroscopic screen moves up to 66″ above floor (vertical).
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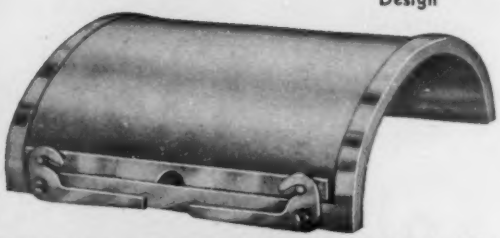

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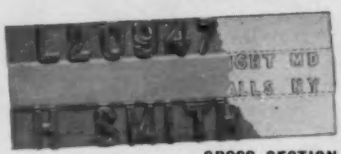

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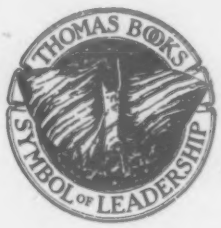

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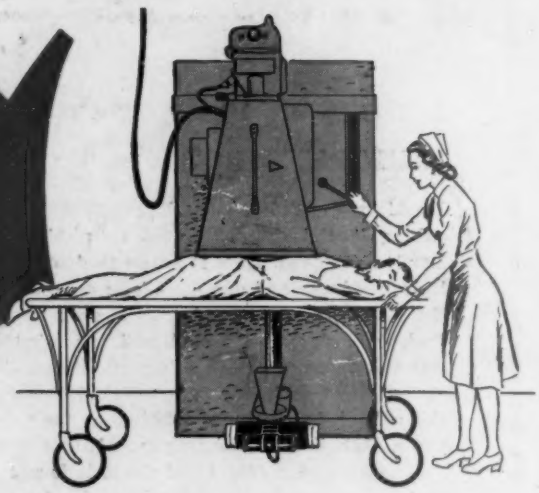

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